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THE GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE

WALTER A. BLOEDORN, M.D.*

Washington, D. C.

FOREWORD

During the 130 years since the School of Medicine of the George Washington University opened, it has received numerous honors, and the faculty regards the opportunity to contribute to this special number of *The American Surgeon* an addition to those honors.

The George Washington University School of Medicine has a long, interesting, and honorable history. It was opened in March 1825. Of the schools of medicine now in existence in the United States, it is the eleventh in chronological order of founding. Plans for the school were formulated, and professors were elected in 1821, four years before the school was opened.

In 1844 the School of Medicine established one of the earliest teaching hospitals in the country, when it petitioned Congress for the use of the Washington Infirmary—a building which had had a varied history of use as a poorhouse, a jail, and an insane asylum.

The Civil War briefly interfered with, but did not completely halt, the activities of the School of Medicine; the Infirmary reverted to the Government at the outbreak of the war and was burned to the ground six months later; one professor left for the South to serve as physician to Jefferson Davis, president of the Confederacy; other faculty members, as well as the medical students, were divided by the controversy. Stability was restored after the Civil War, and thereafter the School of Medicine continued a course of steady, consistent progress, with respect to facilities, faculty, curriculum, and research.

A recent landmark in the history of the school was the opening in 1948 of the new George Washington University Hospital. This splendid hospital, with a capacity of 400 beds, is completely furnished with the latest and most scientific apparatus and equipment. It is staffed and controlled by the faculty of the School

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of Medicine, and provides excellent clinical opportunities for the instruction of medical students, with virtually every specialty in the fields of medicine and surgery assigned space and equipment in both the out-patient department and the bed-patient section. Another important recent addition to the teaching facilities is the George Washington University Cancer Clinic, a modern building opened in 1954, housing the cancer detection clinic and special laboratories for research related to cancer.

Other buildings include the five-story medical school building and an adjacent research building.

Students at the George Washington University School of Medicine receive clinical training at the University Hospital, the University Cancer Clinic, and at five affiliated hospitals. The affiliated hospitals are: District of Columbia General Hospital, Washington's city hospital with 1,400 beds and an out-patient department where more than 75,000 patient-visits are made each year; Walter Reed Army Hospital, one of the Armed Forces' outstanding teaching hospitals; Martinsburg and Mt. Alto Veterans Administration Hospitals; Children's Hospital; St. Elizabeths Hospital, one of the world's most famous mental hospitals, which cares for 7,500 patients with virtually every known psychiatric and neurologic disorder, and which this year is celebrating the one hundredth anniversary of its founding.

The Department of Surgery of the George Washington University School of Medicine is active in the training of medical students, interns, and residents in all these hospitals. Faculty members of the Department of Surgery also serve as consultants to these hospitals, as well as to the new Clinical Center of the National Institutes of Health.

This wide diversity of clinical facilities provides remarkable opportunities for the training of medical students in surgery, and for the training of interns and residents in virtually every subspecialty of surgery, in addition to providing a splendid background in general surgery.

The Department of Surgery conducts several research laboratories and participates in research in surgery in affiliated hospitals.

We wish to thank the publishers of *The American Surgeon* for this opportunity to tell our colleagues throughout the country a little of the past and present of The George Washington University School of Medicine, as well as the opportunity to provide a group of papers from our Department of Surgery, which we hope will be informative and useful to all.

INTRATHORACIC METHOD FOR HYPOTHERMIA; A FOLLOW-UP STUDY*

HOWARD C. PIERPONT, M.D., BRIAN BLADES, M.D.

Washington, D. C.

Interest shown in a simple method for inducing hypothermia reported over a year ago[1] has provided the authors with sufficient stimulus to record this follow-up study.

Perfusion of cold physiologic saline solution into the opened thorax was found to be an effective method for cooling the circulating blood volume. Advantages cited were: (1) direct observation of the heart during cooling, thus reducing the time factor in treating cardiac complications; (2) instantaneous control of the perfusate, allowing immediate cessation of cooling as soon as anoxic electrocardiograph tracings appeared; (3) a rapid, effective cooling technic which eliminated patient handling and bulky equipment; and (4) an equally effective technic for warming, by elevating the temperature of the perfusate.

Survivors of the initial animal experiments now have been killed one year later, and no gross pathologic changes were found at autopsy.

The report of success in one human case[1] now has been expanded to 4 such cases. Although this method of cooling is limited in scope to open chest procedures, we believe these additional cases and the follow-up information indicate intrathoracic cooling is a useful method when used alone or to supplement more popular skin cooling technics.

EXPERIMENTAL METHODS

Adult dogs had fifth left rib resections under pentothal sodium anesthesia. Respiration was maintained by intermittent positive pressure oxygen flow, using a pneophore valve.

Sterile physiologic saline solution, chilled to a temperature of 1 to 3 C. by an iced coil apparatus, then was allowed to flow directly into the open thorax. As the perfusate accumulated, the overflow was siphoned off.

Using rectal temperatures, an empirical limit of 26 C. was selected. In 15 consecutive experiments, the average time required to reach this level was 67.8 minutes. Any shivering was controlled by additional anesthetic.

As soon as cooling was terminated, the animals' temperatures seemed to vary in proportion to the depth of anesthesia. Temperatures tended to drift downward in the more depressed animals and to swing upward in animals who were less deeply anesthetized.

Rewarming the animal was accomplished simply by heating the perfusate to 45 C. In general, this required a third less time than cooling.

* This investigation was supported in part by a research grant (H-142) from the National Institutes of Health, Public Health Service.

From the Department of Surgery, the George Washington University School of Medicine, Washington, D. C.

TABLE I

Intrathoracic hypothermia in fifteen consecutive animal experiments

Animal Number	Survival in Days	Cause of Death
1. 0056	3	Pleural effusion*
2. 0060	4	Pulmonary edema
3. 0053	49 (killed)	—
4. 0059	1 (cardiac arrest)	Myocardial hemorrhage
5. 0057	2	Pleural effusion*
6. 0055	344 (killed)	—
7. 0064	0	Cardiac arrest
8. 0063	33 (killed)	—
9. 0062	342 (killed)	—
10. 0066	502 (killed)	—
11. 000B	349 (killed)	—
12. 0068	355 (killed)	—
13. 0071	0	Cardiac arrest
14. 0069	362 (killed)	—
15. 0089	334 (killed)	—

* Due to break in asepsis.

EXPERIMENTAL RESULTS

Of the original 15 consecutive experiments, 2 animals died from cardiac arrest. A third animal developed cardiac arrest but survived 24 hours after three hours of cardiac massage. At autopsy, the myocardium was hemorrhagic. Two other animals died two and three days after operation from a pleural effusion attributed to breaks in asepsis in the early period of the experiment. The sixth animal died four days later of pulmonary edema or distemper.

The remaining 9 animals survived until killed. One of these developed ventricular fibrillation which readily responded to electro shock therapy. Two of the 9 animals were killed 33 and 49 days later, and no post-mortem changes could be found. The remaining 7 remained in the colony as healthy animals until killed approximately one year later. At autopsy the lungs were healthy and pink, and no adhesions were found (table I).

CLINICAL METHODS

The procedure used in human cases varied more toward simplicity than in the laboratory. Standard liter bottles of physiologic saline solution were cooled overnight by placing 30 to 40 bottles in a clean, watertight, clothes hamper packed with cracked ice from the hospital kitchen. On the morning of the procedure a sterile Valentine bottle was suspended near the patient, allowing the rubber tubing to reach the open chest. Additional help was recruited to keep the Valentine bottle filled. The temperature of the stored saline solution was between 1 and 3 C. This temperature was elevated to 10 to 14 C. by adding saline solution at room temperature.

The stream of perfusate was controlled manually, using a clamp, and it was directed away from the heart. A laboratory thermometer, held in the left hand,

enabled spot checks of the temperatures of the perfusate to be made in all recesses of the open thorax. It is important to note here that the temperature of the fluid bathing the pericardium was kept at 26 C. or above.

Continuous electrocardiograph tracings served as the control for the speed of cooling. Medical consultants constantly studied the oscilloscope tracing or the direct writing electrocardiograph. Any sign of anoxia was the signal to stop the cooling process. Repeatedly this simple maneuver allowed correction of these changes in 8 to 10 cardiac cycles. Only after the tracing returned to its previous status was cooling resumed. In none of these experiences was there a single incident of fibrillation. As soon as anoxic patterns increased in frequency with return to cooling, the temperature was deemed minimal for the patient and the operative procedure was begun.

Rewarming consisted in simply reversing the temperature of the perfusate to 45 C.

In 2 of these patients a small rubber mattress was used as an additional cooling method. The patients lay on the chilled mattress during induction of anesthesia and opening the chest. This same mattress served as a warming element before and during the chest wall closure. The enhanced speed of cooling when both technics were employed is shown graphically in figure 1.

It also is noteworthy to keep the operating room as cold as practical. This simple factor may be overlooked where air conditioning is present.

Rectal temperatures were recorded continuously by thermocouple potentiometers.

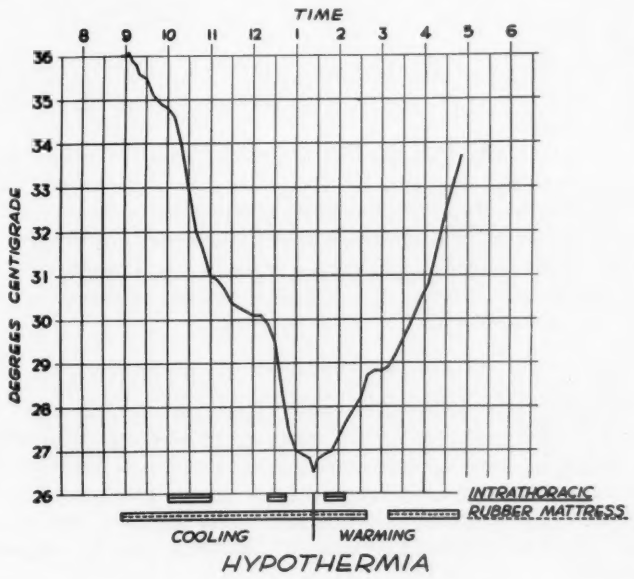


FIG. 1

TABLE II

Intrathoracic hypothermia in four human cases

Age	Minimal Rectal Temperature	Total Cooling Time (Intrathoracic)	Total Warming Time (Intrathoracic)	Adjunct Cooling Methods	Operative Lesions
25	30 C.	155 minutes	115 minutes	none	Saccular Aneurysm (Aortic Arch near Subclavian branch)
52	26.5 C.	85 minutes	30 minutes	ice mattress	Saccular Aneurysm (Innominate Artery and Aortic Arch)
48	27 C.	none	60 ± minutes	ice mattress	Saccular Aneurysm (Aortic Arch involving all branches)
61	32.5 C.	85 minutes	60 ± minutes	none	Saccular Aneurysm (Homologous Vessel Graft of Descending Thoracic Aorta)

CLINICAL RESULTS

The depth of hypothermia, the rate of cooling and the adjunct cooling methods in 4 human cases is itemized in table II. The youngest of these patients was 25 years of age and the oldest was 61.

Ventricular or auricular fibrillation did not occur. The anoxic pattern on electrocardiography reached an alarming frequency at 32.5 C. in the 61 year old woman, whereas this did not occur until the temperature dropped to 30 C. in the 25 year old man.

Three of the patients are alive and well, without roentgenographic evidence of pulmonary changes, 5 to 14 months after operative procedures employing this method for hypothermia.

The fourth patient died 12 hours after operation from some embolic phenomenon. Hypothermia was produced by the chilled mattress technic. The intrathoracic method was unnecessary as dissection of a large aortic aneurysm was so time consuming, the body temperature reached a desired level by skin cooling alone. Intrathoracic warming was employed, however. Hypothermia can not be included as a cause of death. Surgical details in this procedure are reported separately[3] as are those of the 61 year old woman.[4]

DISCUSSION

The complex changes induced in animal physiology by altering the single factor of temperature are unfolding gradually. Many concepts remain hypothetic.

Rapid cooling has been considered desirable. From a practical viewpoint this is easily understood. Long operative procedures become even more lengthy when hypothermia is employed. Stress phenomena must certainly become involved proportionately. The benefits of hypothermia *protection* must be weighed against this time factor.

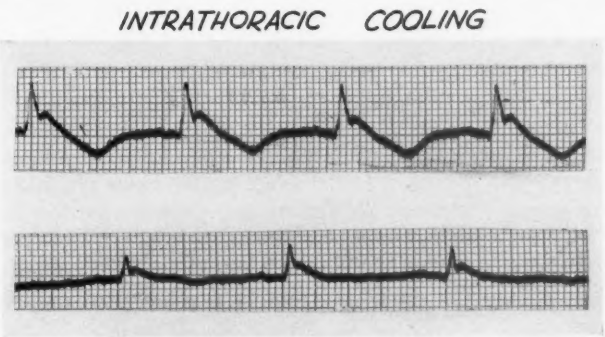
Age has been considered by some investigators a contraindication to the use of hypothermia. Our experience in one 61 year old patient would indicate the degree of cooling is more limited. However, the rate of cooling (1 C. per 19 minutes) was more rapid than that in the 25 year old patient (1 C. per 26 minutes) We can only postulate that a lower temperature might have been reached safely if more time had been taken. This raises a question regarding the desirability for speed in cooling older patients.

Ventricular fibrillation is well known as the leading complication in hypothermia. The most practical way to detect this impending disaster is by constant electrocardiograph monitoring. Lange, Weiner and Gold[5], have described the *anoxic patterns* found in hypothermia. Electrocardiograph tracings in our human cases followed the same changes (fig. 2).

In vitro studies of the shifting oxyhemoglobin dissociation curves for blood at subnormal temperatures were known to Brown and Hill[2] and have recently been confirmed in vivo by Penrod.[6] Just how blood oxygen dissociation is affected in living tissue under hypothermia may be related to the individual capillary temperature of the tissue in question. Additional protection may be necessary to conserve the intrinsic myocardial temperature. Some investigators insulate or even warm the pericardium while cooling. During hypothermia the preservation of oxygen utilization in the myocardium as shown by unaltered arteriovenous differences[6, 7] indicates a wide margin of safety peculiar to the heart.

Sudden alterations in circulating blood PH are believed to be closely related to ventricular fibrillation. The release of cardiac inflow occlusion occasionally precedes cardiac irregularities in our laboratory experiences. We are at present attempting to measure these sudden changes in relation to fibrillation.

The influence of blood PH was demonstrated clinically in the first human case. While rewarming the patient, electrocardiograph changes were announced as indicating anoxia. The anesthesiologist agreed to stop manual lung inflations for



ST-T WAVE CHANGES (LEAD II)
SHOWING ANOXIC PATTERN
(RECTAL TEMP. 29° CENT.)

FIG. 2

several minutes. The medical consultant soon thereafter reported an improvement in the tracings. We can explain this only by a temporary accumulation of CO_2 in the circulating blood. It does correspond to Lange, Weiner and Gold's[5] report of the beneficial effect of lowering the blood PH in the experimental animal under hypothermia.

SUMMARY AND CONCLUSIONS

A simple technic for producing hypothermia is presented.

The original experimental cases are reviewed after one year of study.

Four adult human cases, using this technic, are presented. No cardiac complications occurred which can be attributed to the method.

One of the 4 human cases presented is that of a 61 year old woman. This experience of hypothermia in the older age group is discussed.

With a method which offers control of myocardial anoxia and immediate treatment of ventricular fibrillation should it occur, the dangers of hypothermia in adults and older age groups may be reduced.

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THE ANTICOAGULANT EFFECTS OF HEPARIN AND PHOSPHORYLATED HESPERIDIN SINGLY AND IN COMBINATION*

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In the course of a search for *antilipemic* agents related to heparin our attention was directed to phosphorylated hesperidin. This drug is a flavanoid polysaccharide, similar to rutin, which shares certain properties with heparin.[1] As reported by Beiler and associates[2] phosphorylated hesperidin did not effect blood clotting either in vitro or in vivo in animals. A more recent investigation by Bourgain[3] and associates has shown that intravenous administration induced significant prolongation of the clotting time and increased the titers of antithrombin in rabbits. The present report summarizes observations in human beings of the effects of heparin and phosphorylated hesperidin, and their combination, upon the silicon clotting time and prothrombin time.

METHODS

Equipment was siliconized using silicon solution prepared from General Electric Dri-film #9987 diluted to 5–10 per cent with acetone. This solution was poured into and through clean, dry, 10 cc. syringes and 10 mm. by 75 mm. test tubes, all inner surfaces being left in contact with silicon for several minutes. The glassware then was rinsed twice with distilled water. This fixes silicon to glass. It should be done under a hood because of the development of pungent fumes. Syringes and tubes then were rinsed repeatedly under running tap water and dried in a hot oven. The venipuncture needles, sharp no. 20, were not coated because silicon adheres poorly to metal. The glassware was resiliconized after each use.

With the rare exceptions to be mentioned, only a prompt, clean, venipuncture was accepted to keep contamination by subcutaneous fluid at a minimum. For the same reason a total of 7 cc. of blood was withdrawn at each puncture and the initial 5 cc. were discarded.[4]

Clotting times were done employing 2 tubes, each containing 1 cc. of whole blood, and kept in a water bath at 37 C. Tube I was gently tilted every 5 minutes until partial clotting occurred, then both tubes were tilted every 5 minutes until completely clotted. The end-point was established by the absence of flow in the clot with the tube inverted and gently tapped. After some preliminary practice, determination of the end-point presented no particular problem. All readings,

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This study was supported in part by a grant from The Washington Heart Association, Washington, D. C.

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TABLE I

Summary of silicon clotting times in 9 subjects

Subject	Tube	Control	10 mg. Heparin I.V.		Control	10 mg. Heparin + 200 mg. Hesperidin	
			1 hr.	1½ hr.		1 hr.	1½ hr.
J.C.	1	25	27	8	28	47	38
	2	28	35	14*	33	81	40
W.S.	1	30	29	17	32	17	41
	2	32	39	26	34	52	56
A.W.	1	27	55	26	29	64	38
	2	31	36	31	36	104	82
T.K.	1	42	66	65	24	132	47*
	2	42	69	59	36	157	66
R.M.	1	26	107	75	39	128	220
	2	48	192	102	40	292	212
H.C.	1	32	25	25	33	143	95
	2	30	55	58	33	153	70
W.C.	1	42	52	33	24	67	39
	2	45	59	39	39	122	44
C.I.	1	20	48	11*	25	119	65
	2	20*	64	16	37	148	85
M.S.	1	59	119	108	63	245	225
	2	63	150	111	64	266	260

* Difficulty with venipuncture.

with a few exceptions, were done by the same two observers. Clotting time of tube II was taken as the final reading. Infrequently tube II was found to be the first to clot and in such instances the tube I reading was taken as the end-point.

The study was divided into two parts. In both parts the subjects studied were in the postabsorptive state and included healthy hospital personnel and patients free of primary liver disease. In part one, using 9 subjects, we were interested in determining the effects of the simultaneous administration of heparin and phosphorylated hesperidin* intravenously. A dose of heparin was selected which produced slight to moderate prolongation of the clotting time at 60 minutes.

The study was made as follows: after a control blood sample was obtained, 10 mg. of aqueous heparin were given intravenously, and samples were taken at 60 and 90 minutes. As each sample was obtained, the blood was immediately placed in its tube in the water bath and testing for clotting were initiated. On the following day, the procedure was repeated with 200 mg. (2 cc.) of phosphorylated hesperidin and 10 mg. of heparin given intravenously in the same syringe mixed with 14 cc. of normal saline solution.

The results are summarized in table I and represented graphically in figure I. It can be seen from the data that control values compare quite closely in the 9 individuals and in the same individual on consecutive days. Using heparin alone, there was minimal prolongation of the clotting time at 60 minutes, and at 90 minutes most clotting times had returned to control values. With the heparin-

* Supplied through the courtesy of Mr. W. S. Swain, National Drug Co., Philadelphia, Penn.

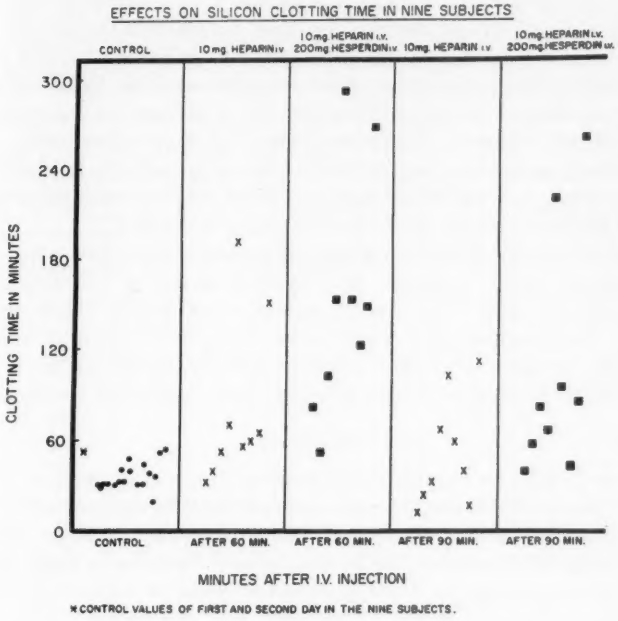

FIG. 1

TABLE II

Silicon clotting time and prothrombin time in 6 subjects after 200 mg. phosphorylated hesperidin intravenously

Subject	Tube	Control	Clotting Time		Prothrombin Time	
			60 minutes	90 minutes	Control	60 minutes
E.C.	1	33	19*	35	61%	30%
	2	44	24	38		
J.A.	1	45	42	65	50%	30%
	2	53	58	60		
P.S.	1	29*	42	30	100%	50%
	2	27	46	31		
S.B.	1	33	48	60	64%	33%
	2	33	51	60		
E.F.	1	44	137	122	64%	33%
	2	69	93	97		
L.S.	1	35	57	51	86%	35%
	2	35	49	51		

* Difficulty with venipuncture.

hesperidin combination, there was prolongation of the coagulation time over that of heparin alone at 60 minutes and the effect persisted at 90 minutes.

In part two, 6 individuals were given 200 mg. of phosphorylated hesperidin intravenously, diluted with 15 cc. of normal saline solution. In all other respects the same procedure was followed as in part one of the study. In addition, the one stage prothrombin time was determined before and 60 minutes after the injection. The results of the silicon clotting time are shown in table II. It is seen that the clotting time was moderately prolonged in 3 of the subjects, while the prothrombin time was slightly increased in all 6 of the subjects (table II).

To evaluate the influence of oral administration of phosphorylated hesperidin on prothrombin time, 5 subjects were studied, including 3 healthy physicians and 2 patients, in all of whom control one stage prothrombin times were normal. The drug was given in equally divided doses four times daily after meals and at bed time in amounts from 1200 to 2400 mg. per day, for periods up to one week. In none of the subjects was there any significant increase in prothrombin time.

COMMENT

This study confirms in man the prolonging effects of intravenously administered phosphorylated hesperidin upon the clotting time as reported by Bourgain and associates in animals. These authors found no change in the prothrombin time of the rabbit whereas in our human subjects there was a slight but definite increase. The source of this discrepancy is not readily apparent and will require additional evaluation. On the other hand, the prothrombin time was uninfluenced by oral dosage with hesperidin under the conditions of our experiment.

Our data also appears to show that phosphorylated hesperidin potentiates the effect of heparin upon blood clotting both in the degree and duration of the prolongation of coagulation. Since heparin, as a biological, is difficult and expensive to prepare, chemically formulated phosphorylated hesperidin may have clinical utility. However, its place in the practice of anticoagulant therapy in patients remains to be determined. Bourgain and associates found that intravenous doses of 200 mg./Kg. proved to be promptly fatal in the rabbit with autopsy showing extensive hemorrhages in the spleen, lungs, and myocardium. In our human subjects, employing doses only a fraction of these, we have seen no instances of hemorrhagic complications or other significant side effects.

SUMMARY AND CONCLUSIONS

A study has been conducted in man of the effects of intravenously administered heparin and phosphorylated hesperidin, singly and in combination, upon the silicon clotting time and prothrombin time. It was found that intravenously administered phosphorylated hesperidin alone produced slight prolongation of the clotting and prothrombin times. In combination with heparin, it potentiated the effect of this drug upon the clotting time. Phosphorylated hesperidin appears to represent a heparin type anticoagulant the clinical utility of which will have to be determined by further evaluation.

ACKNOWLEDGEMENT

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FAMILIAL OCCURRENCE OF THROMBOSIS OF THE TERMINAL AORTA

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The recently developed technics for the preservation of arterial homografts have stimulated much interest in the problem of segmental obstruction of the abdominal aorta and its major branches. Such occlusions develop insidiously in patients with localized arteriosclerotic plaques and thrombosis of the small residual lumen. In the past treatment has consisted of vasodilating drugs, Buerger's exercises, sympathectomy, arteriectomy and more recently thromboendarterectomy.[4, 6, 7, 11, 15, 17, 18] Patients with localized occlusion of the lower aorta and iliacs and patent femoral vessels now can be offered resection of the occluded area with replacement by bifurcation grafts.[2, 3, 5, 9, 13]

Even though it has been demonstrated that arterial homografts merely act as a framework for the development of a new vessel wall, and[14, 16] that calcification of the graft and aneurysm formation in the grafted vessel above and below the graft have been described,[12] the prognosis of untreated persons with this condition is so uniformly poor that grafting is warranted.

It has been generally accepted that severe forms of arteriosclerosis may show a certain familial predilection for specific sites,[1] but we have not found any reports of the familial incidence of thrombosis of the terminal aorta. Hence, it appeared worthwhile to report its occurrence in three males, two brothers and a cousin. Further investigation has shown that the two brothers had a sister who had bilateral amputations for gangrene of the feet and since has died. A maternal uncle had a unilateral amputation for a similar condition. The cousin has a brother whose leg was amputated for arterial insufficiency and his father apparently died of gangrene. This familial relationship is shown in figure 1.

These patients emphasize the severity of the disease as well as its progressive and fatal nature. Many recent reports on aortic thrombosis deal with the happy results of thromboendarterectomy or grafting.

CASE REPORTS

The following cases illustrate what the outcome may be if the condition is not recognized and treated early.

Case 1. S. C., a 61 year old handyman, first was admitted to this hospital in 1947 for treatment of pulmonary tuberculosis. He complained of claudication and rest pain in his calves since 1940. In 1947 he was diagnosed as having varicose veins of the left leg, which were ligated in 1948. Postoperatively, he complained bitterly of pain in the right leg. This was only partially relieved by sympathetic block. Medical and surgical consultants thought that sympathectomy would be of no value. Moreover they believed that this was *ischemic*

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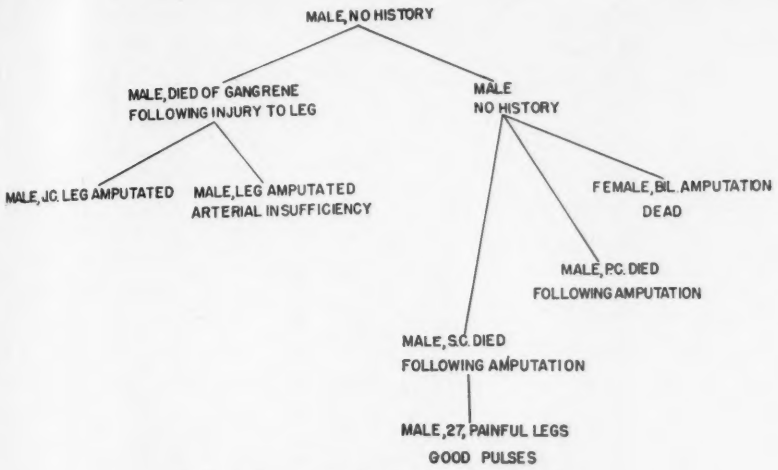

FIG. 1. Chart showing incidence in C family

neuritis, unaware of a more serious disease in the aorta. On medical therapy there was sufficient improvement to warrant discharge from this hospital with frequent check-ups. He again was admitted on Aug. 3, 1953 complaining of claudication of both legs, more severe in the right. Physical examination at this time showed a thin, white man, who did not appear acutely ill. The heart and lungs were essentially normal. the blood pressure in the arm was 180/110. Scars of right thoracoplasty, right saphenous vein ligation and appendectomy were well healed. It was noticed at this time that erythema developed in the dependent position and that there was blanching on elevation of both legs. Dorsalis pedis and posterior tibial pulses could not be felt on the right and were faint on the left. Neurologic examination was negative. On August 5, an epidural block showed an average elevation of skin temperature approximately 3 to 5 degrees in the toes but above the toes no appreciable elevation of temperature. Examination of the major vessels again showed that the pulsations on the left were faint, but were more prominent than the pulsations on the right side, i.e., femoral pulsations were unequal as were the popliteal, dorsalis pedis and posterior tibial. Oscillometry showed marked reduction in excursions for the left leg as compared with the right. On August 10, a percutaneous right femoral arteriogram was done which revealed a fair visualization of the arterial system of the thigh (fig. 2) and leg. There was no evidence of block nor segmental narrowing of these vessels. Immediately following this, the patient developed an intense pain in the left leg with a decrease in all pulsations. The leg was cold, blanched and extremely painful. There was loss of sensation, and it was thought that acute arterial occlusion in the left leg had occurred. Immediate epidural block was done with instillation of local anesthetic through the epidural tube, but without relief of symptoms. On August 21, translumbar aortography was done, (fig. 3) which showed irregular filling of the aorta with occlusion from just below the renals down to the bifurcation. There was no evidence of collateral circulation. The roentgenologic interpretation was thrombosis of the lower aorta.

The foot did not improve and the patient complained of increasing pain. On August 27, exploration of the left femoral artery showed complete absence of blood in the narrow but patent vessel. Abdominal exploration showed no pulsation in the aorta from a point just below the renal vessel downward. The aorta was dissected from surrounding tissue that had undergone intense perivascular reaction, and was controlled by clamps. The bifurcation was opened and the incision was extended down into the left iliac artery. Both the

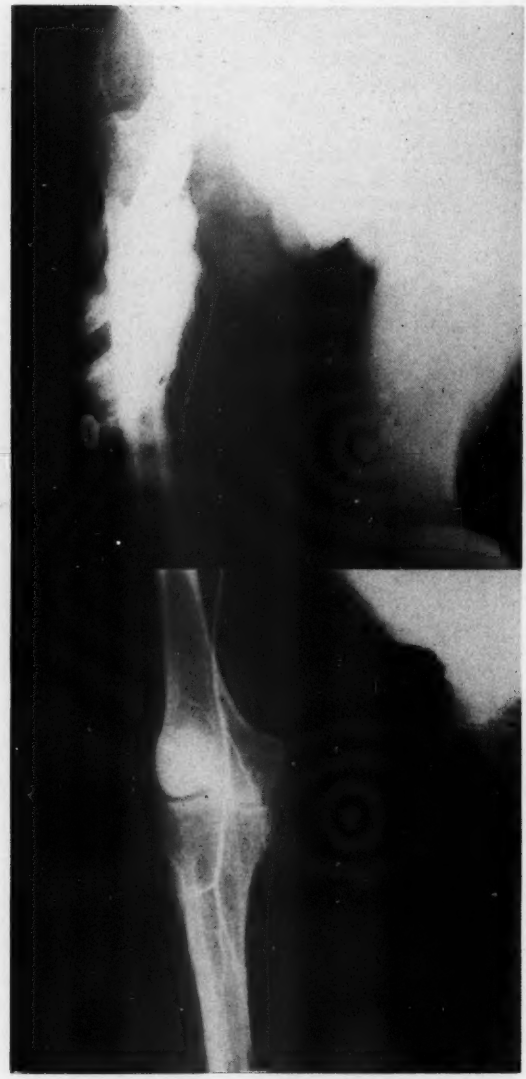

FIG. 2. Right femoral arteriogram (composite) of S.C.

aorta and the left iliac artery were completely occluded by grumous material, which extended up the aorta, above the level of the renals. By gentle probing and repeated aspiration it was possible to remove some of the occluding material until a faint trickle of blood was obtained. Both iliac vessels were cleaned out in the same manner until some back flow of blood was obtained. However, the force of the stream was very weak and no hope was

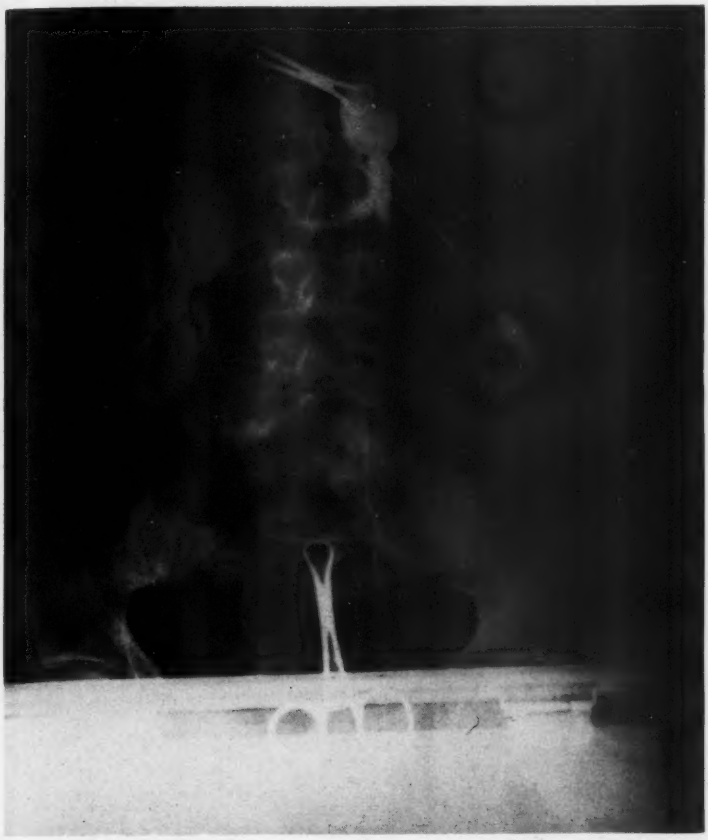

FIG. 3. Translumbar aortogram of S.C.; note level of occlusion at renal arteries

held for an adequate or sustained circulation. Bilateral sympathectomy was done. The vessel then was closed with arterial sutures. Following this procedure the left leg remained cold and purple below the knee and the patient continued to have severe pain. Necrosis and ulceration ensued. On September 2, an amputation was done at the middle of the left femur with loose skin flaps over the bony end. The wound did not heal and over a period of months the patient gradually became worse. Superficial pressure sores were noted over the sacrum and bony prominences bilaterally until eventually there was frank ulceration and necrosis of these areas. Revision of the amputation stump was attempted on September. During this operative procedure it was noted that there was very little blood supply to the skin or muscles. The patient became rather delirious and complained of severe pain. Adequate doses of demerol did not relieve him and a trial administration of chlorpromazine was given with satisfactory sedation. Doses of demerol were decreased and the patient was temporarily relieved. Within a short time pain again became uncontrollable and on October 15, because of severe pain, a posterior laminectomy was done with cordotomy on the left. This afforded good relief of pain in the left leg. The patient began to complain

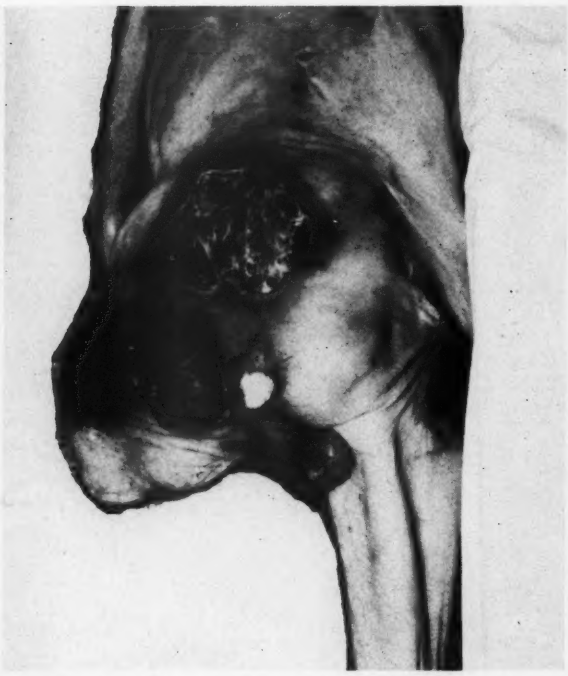

FIG. 4. Postmortem view of sacral decubiti, ulcerations over right and left trochanters and left ischial tuberosity.

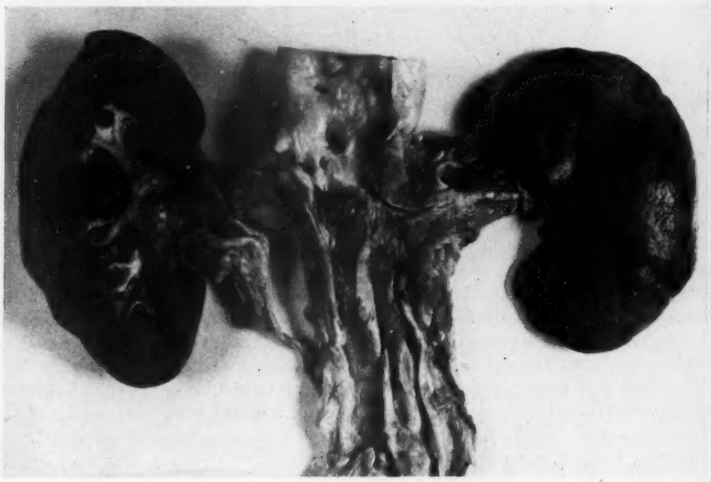

FIG. 5. Postmortem specimen of aorta and bifurcation. Thrombus descends from level of renal arteries involving bifurcation. Renal arteries were proved to be patent.

of pain on the right side and again required moderate doses of demerol and chlorpromazine. The patient's course was generally downhill with continued necrosis and infection of the stump and rapid enlargement of decubiti (fig. 4). He died on Dec. 6, 1953.

Autopsy showed thrombosis of the aorta at the level of the renals extending down into the major vessels of the leg (fig. 5).

This history is given in detail to empahsize a point made by Leriche[10] concerning such patients in whom the disorder "appears to be a disease with a long course presenting, for a long period, symptoms which have no meaning to the physicians, unpleasant as they may be for the patient". Some of these cases have been labeled varicose veins, or polyneuritis.

Case 2. P. C. was a 63 year old white male farmer. He was a brother of S. C. and first cousin of J. C. He was admitted to the hospital in January 1953 with a four year history

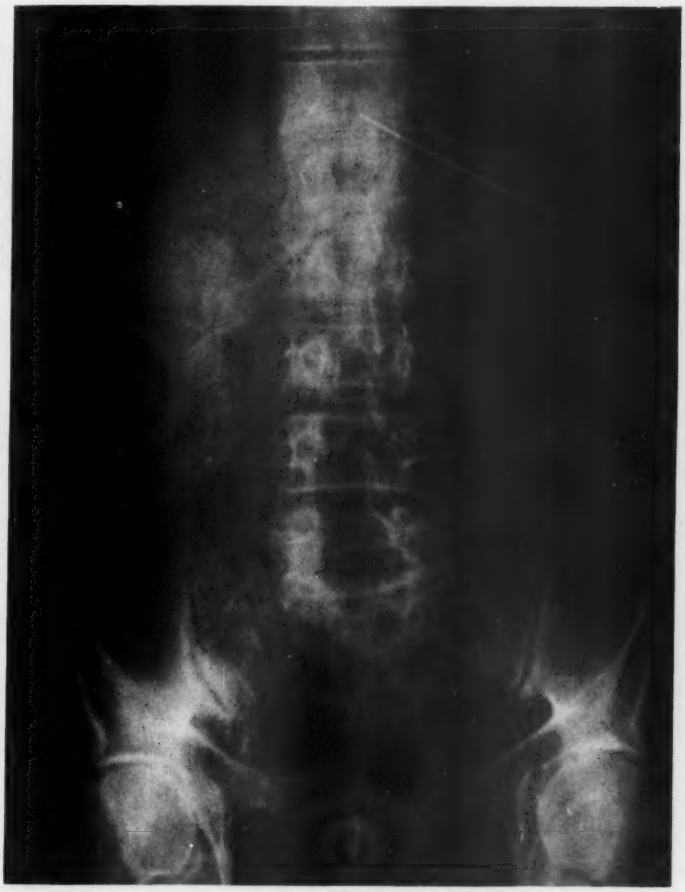

FIG. 6. Translumbar aortogram of P.C., showing occlusion at level of the third lumbar vertebra.

of a burning pain in the right foot and claudication. In 1949 he had a left lumbar sympathectomy for similar pain in the left foot with excellent results. At the time of admission he had only mild cramps in the left calf since sympathectomy.

Physical examination essentially was negative except for the lower extremities. The right toes were cool and reddened. The skin of the left foot appeared normal. Both femoral pulsations were weak, as were the popliteal and dorsalis pedis. The posterior tibials were not palpable. On January 22, a right lumbar sympathectomy was done. There was no improvement in the temperature of his foot, although there was some subjective improvement. He returned to the hospital in March complaining that for six seeks the right foot had been cold and painful. Examination showed that the right leg was cool up to the knee with pallor on elevation and cyanosis when dependent. The skin was smooth and shiny. A translumbar aortogram showed occlusion of (fig. 6) the aorta at the level of the third lumbar vertebra with questionable filling of the left common iliac artery. Midthigh amputation on the right became necessary in May because of pain of the foot and beginning necrosis. The femoral artery, although arteriosclerotic, was patent. The wound failed to heal and the stump was reamputated three weeks later. Necrosis of the stump recurred and finally in October disarticulation of the hip joint was done. A portion of the wound again became necrotic and infected and a sacral decubitus rapidly enlarged. The patient died rather suddenly on Dec. 19, 1953.

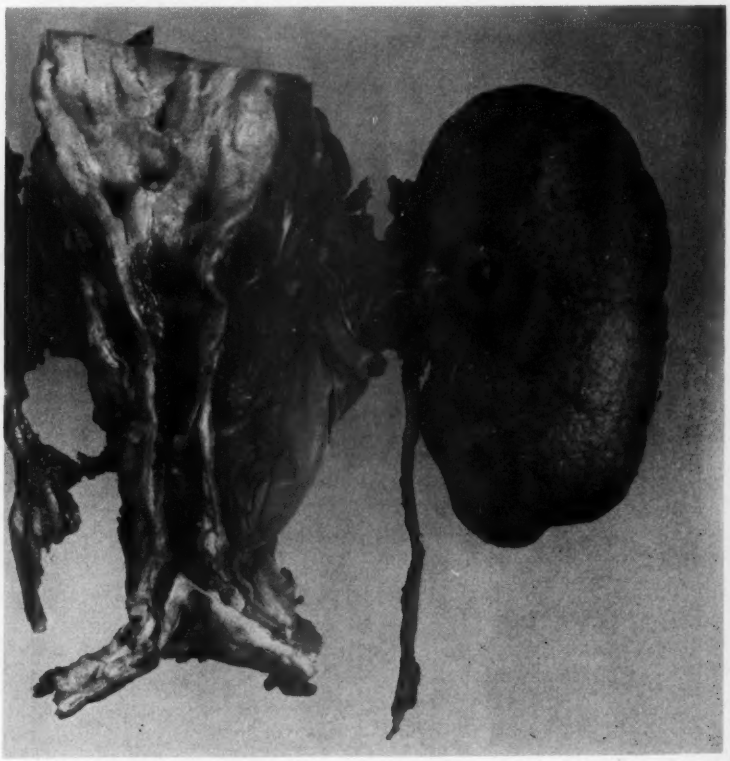

FIG. 7. Postmortem specimen of P.C.; note thrombus blocking aorta and bifurcation from level of the third lumbar vertebra.

Autopsy showed occlusion of both common iliacs and of the aorta up to the level of the inferior mesenteric. There was slight arteriolar nephrosclerosis (fig. 7).

Case 3. J. C., a 67 year old white male farmer, first cousin of S. C. and P. C., first seen in September 1952 with complaint of throbbing pain in the left great toe. During the winter of 1951 the toes of the left foot had been colder than the right. In June 1952 pain, especially at rest, began in the left great toe. On walking he noted a tired feeling in the leg and foot but less pain in the toe.

Physical examination showed a blood pressure of 154/90, a slight systolic murmer and a right inguinal hernia. The left leg was cool from midleg down and there was dry gangrene of the left great toe. The dorsalis pedis, posterior tibial and popliteal pulsations were absent on the left and the femoral was diminished. On the right, all pulses were palpable. Sympathetic block by bilateral epidural technic showed some rise in temperature on the right but less than a degree increase on the left. Oscillometry after block showed no pulsations in the left ankle and calf and only a swing of 2 in the thigh. On September 2, bilateral lumbar sympathectomy was done with considerable improvement in the right foot. The left great toe showed progression of gangrene and was amputated at the metatarsophalangeal joint on September 25. The wound failed to heal, was extremely painful, and formed an ischemic ulcer. On December 18, midthigh amputation was done. It healed satisfactorily, but in March 1953 an episode of thrombophlebitis occurred in the stump. Until recently he was ambulatory with a prosthesis and on limited activity had only a tired feeling in the right instep. The pulses in the remaining leg were present although the posterior tibial was diminished. On Jan. 2, 1954, sudden pain in the anterior calf and dorsum of the foot occurred, and the peripheral pulses distal to the femoral disappeared. On anticoagulant therapy the pain disappeared and he was able to ambulate for short distances, but no pulsation could be felt distal to the femoral.

COMMENT

In the past the treatment for aortic occlusion can be described as *too little and too late*. Wylie and others[4, 6, 15, 17, 18] have advocated thromboendarterectomy, but this is subject to recurrent thrombosis because the remaining vessel lacks an intimal lining. Leriche[10] in 1940 first described the syndrome of thrombotic obliteration of the inferior part of the abdominal aorta. To check the advance of the disease and to remove the stimulus of the vasoconstricting impulses, he[10] recommended the resection of the thrombosed segment and bifurcation with bilateral sympathectomy. Success of this therapy depends on the development of an adequate collateral circulation. It seems quite clear that the best treatment now available is resection of the diseased aorta and replacement with a homograft.

The sudden development in case 1 of complete thrombosis on the left following femoral arteriography on the right serves to emphasize Leriche's warning that aortography may be dangerous in patients with impending gangrene in whom even a minor degree of vasospasm may be catastrophic.

These patients were all somewhat older than the ones described by Leriche. However, other authors[2, 3, 5, 9, 13] recently have described successful resection and grafting in older people. We recently were able to resect the lower aorta and iliac arteries down to the femorals in a 42 year old man. This was replaced with a freeze dried graft. A previously attempted thromboendarterectomy on the right common iliac had resulted in thrombosis. It is hoped that in the future, the prolonged and painful illness and ultimate death of patients such as S. C. and P. C. may be prevented.

SUMMARY

Three cases of thrombosis of the terminal aorta are presented. These occurred in two brothers and a cousin. A history of arterial disease with amputations in four other members of the family was obtained.

ADDENDUM

Since this paper was submitted for publication, the brother of J. C., who is mentioned as having had an amputation for arterial insufficiency, has been admitted to this hospital. In 1942 the right leg was amputated below the knee for what he was told was Buerger's disease. At present he has severe pain in the left calf, suggesting ischemic neuritis, and absent pulses in the left lower extremity. Aortagram demonstrates a complete block of the left common iliac at the bifurcation of the aorta. Grafting is contemplated if potency of the femoral artery in the thigh can be established.

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THE DIAGNOSIS AND MANAGEMENT OF ESOPHAGEAL PERFORATIONS

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Perforation of the esophagus, either by disease, instrumentation or foreign body is a surgical emergency.[1, 2, 6, 11, 22] If untreated, it carries a grave prognosis. Fortunately, esophageal perforation is not common, but its relative rarity makes diagnosis and treatment no less exacting. Since the most successful results may be obtained by immediate recognition and definitive therapy, it is thought worthwhile to review the causes, diagnostic features and treatment of this catastrophe.

ETIOLOGY

1. *Instrumentation:* Probably the most common cause of acute suppurative mediastinitis at the present time is esophageal perforation by rigid or semirigid instruments. These include the esophagoscope and various types of dilators which are used in the treatment of esophageal strictures. The majority of perforations resulting from esophagoscopy are in the cervical esophagus. Here the posterior wall of the esophagus may be easily torn at the level of the cricopharyngeus muscle, when the instrument is passed without adequate muscular relaxation or when any force is used during a difficult endoscopy.[9]

Perforation by instrumentation at lower levels in the esophagus usually is associated with underlying esophageal disease, such as carcinoma or stricture. When dilatation or forceful examination is done, the esophageal wall may be readily perforated just above the level of the obstructing lesion.

2. *Foreign Bodies:* A sharp metallic or bony foreign body may easily become imbedded in the esophageal wall at any level. This may produce a sudden tear or gradual erosion of the esophageal wall.[21] Examples of the more common foreign bodies are chicken or fish bones, safety pins or small dentures.

3. *Spontaneous Perforation:* This has become a well recognized disease entity in recent years.[8, 12, 13, 19] A large tear in a previously normal esophagus, produced by sudden pressure changes is a catastrophic occurrence. The mortality rate is high.

4. *Perforation Following Chemical Burns:* Severe damage to the esophageal wall may result from the ingestion of lye or other strong chemical agents. The acute disease process may be so severe that erosion and perforation of the esophagus may occur. This may be at any level and also is associated with a high mortality rate.

5. *Other Causes:* Keifer[10] has described acute suppurative mediastinitis resulting from rupture of an esophageal diverticulum, carcinoma, or necrosis of the esophagus from external pressure by aneurysm or mediastinal tumor. These are exceedingly rare.

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SURGICAL ANATOMY

In order to understand the physiology and rationale of treatment of a mediastinitis resulting from esophageal perforation, a knowledge of the fascial planes of the neck and mediastinum is necessary. Numerous studies of these fascial compartments have been made, notably those of Coller and Yglesias,[5] and Pearse.[16]

The most important compartment in a consideration of mediastinal suppuration is the viscero-vascular compartment. This contains the pharynx, larynx, trachea, esophagus, thyroid and thymus glands, nerves, and great vessels. The compartment extends from the base of the skull to about the level of the sixth dorsal vertebra, where it ends by fusion of fascial structures about the bifurcation of the trachea, the aorta, and the pericardium. Within this compartment are found the actual or potential spaces of importance in most mediastinal infections.

1. *Visceral Space:* The area bounded by the pretracheal fascia in front, carotid sheath laterally and buccopharyngeal fascia behind, and containing the trachea, esophagus, thyroid gland and nerves is termed the visceral space. The pretracheal fascia forming the anterior boundary of this space extends from the hyoid bone to the pericardium. Posteriorly, this same layer is continued behind the pharynx and esophagus as the buccopharyngeal fascia. The pretracheal fascia envelopes the thyroid and extends laterally to join the carotid sheath (figs. 1 and 2).

The pretracheal space, lying between the posterior leaf of the pretracheal fascia and the trachea, extends from the larynx to the bifurcation of the trachea. In the mediastinum this space lies between the trachea and the great vessels. It is in this space that infections following tracheotomy or thyroidectomy are found. Drainage of this space is best accomplished through the suprasternal notch.

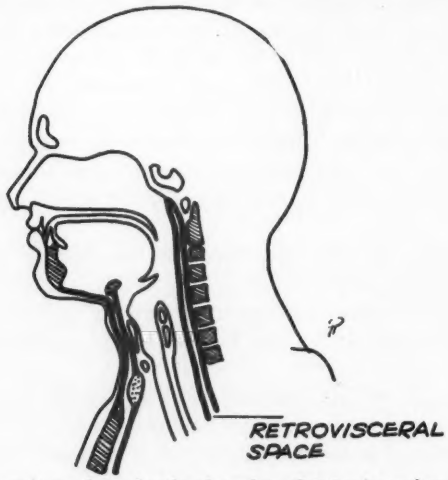

FIG. 1. Diagrammatic section, showing location of retrovisceral space. Note that space is posterior to esophagus and in front of prevertebral fascia. (Courtesy of Dr. Philip Thorek, "Anatomy in Surgery", J. B. Lippincott Company, Philadelphia, Pa.)

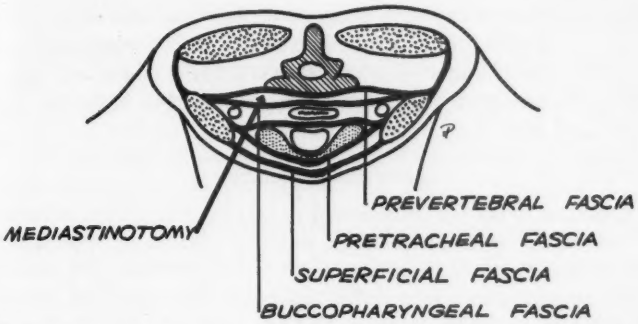

FIG. 2. Cross section of neck, showing fascial planes and route to retrovisceral space by cervical mediastinotomy. (Courtesy Dr. Philip Thorek, "Anatomy in Surgery", J. B. Lippincott Company, Philadelphia, Pa.)

2. *The Previsceral Space:* This is mentioned only to indicate that this space lies beneath the strap muscles of the neck and superficial to the pretracheal space. Its lower limit is a strong attachment of the pretracheal fascia to the undersurface of the sternum, and thus infections of the previsceral space are prevented from descending into the mediastinum.

3. *Retrovisceral Space:* This space lies behind the pharynx and esophagus, and is the most important space in a consideration of esophageal perforations. It also is the most important pathway of infection between the neck and the mediastinum. The retrovisceral space is bounded anteriorly by the buccopharyngeal fascia, laterally by the carotid sheath, and posteriorly by the prevertebral fascia. It extends from the base of the skull to the tracheal bifurcation.[11] Any perforation of the posterior wall of the upper third of the esophagus or the pharynx readily penetrates the thin layer of buccopharyngeal fascia and contaminates the retrovisceral space.

DIAGNOSTIC FEATURES

1. *History:* In the vast majority of cases a diagnosis of esophageal perforation can be made within a short period after its occurrence. Most perforations which occur as a result of esophagoscopy will be recognized by the endoscopist. This may not always be true, however, and the possibility of unrecognized perforation should be constantly kept in mind and each patient should be checked postoperatively for the signs of perforation. History of recent dilatation of the esophagus or ingestion of a foreign body or strong chemical strongly suggest the possibility of perforation if the patient develops chest or neck pain, dysphagia, or fever. History of pre-existing dysphagia and weight loss, with sudden chest pain or respiratory difficulty may suggest perforation of an advanced esophageal carcinoma, frequently producing a tracheoesophageal fistula.

2. *Physical Signs:* The outstanding physical signs produced by an esophageal perforation are those of an acute mediastinitis.[15] These are cervical tenderness and pain on flexion of the neck, crepitation of the soft tissues of the neck, and dysphagia. As the process progresses, fever, tachycardia, and tachypnea will

develop. Should the mediastinitis progress further, the trachea may be pushed forward and tenderness may be elicited on motion of the trachea. There may be widening of the mediastinum on physical examination and other signs of acute suppuration. Spontaneous rupture may be considered as a disease entity, and will be discussed later.

RADIOLOGICAL FEATURES

Little difficulty should be encountered in making a diagnosis of perforation of the cervical esophagus on the basis of physical signs alone. Perforation of the intrathoracic esophagus may not be as clearcut, however, and roentgenologic studies are necessary in making this diagnosis. The principal roentgenologic features of esophageal perforation are:[15]

1. Widening of the mediastinum (fig. 3).
2. The presence of an air-fluid level in the mediastinum.
3. Roentgenologic evidence of air in the soft tissues of the neck or mediastinum.
4. Lateral roentgenograms showing widening of the retrotracheal space, usually with fluid levels.

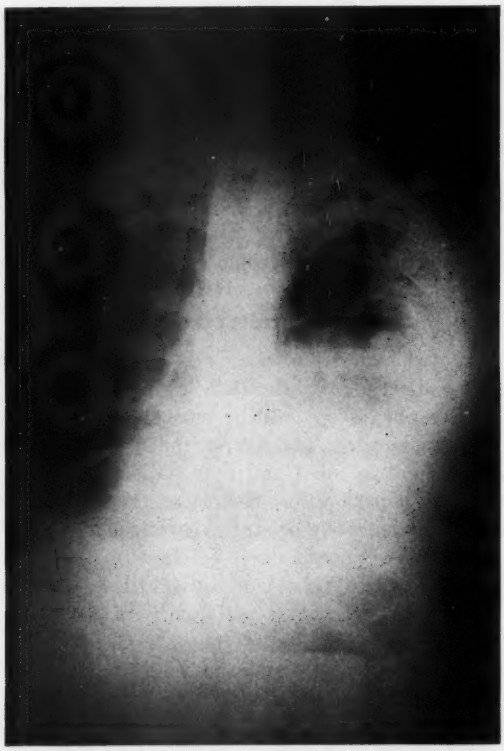

FIG. 3. Case 1, Showing mediastinal emphysema and left hydropneumothorax, 18 hours after rupture of esophagus.

5. Lipiodol swallow. In doubtful cases, or in recognized perforations, where the level of the tear must be determined, a swallow of 20 to 30 milliliters of lipiodol may be given. Roentgenograms taken immediately after this will show the presence of the iodized oil in the mediastinum at or near the level of the perforation.

Children who refuse to take the oil may be fed a mixture of lipiodol and ice cream, rendering it more appealing.[4]

SPONTANEOUS RUPTURE OF THE ESOPHAGUS

This entity has received more frequent mention in the literature of recent years.[8, 12, 13, 19] All cases reported up to 1946 were fatal,[2] but a better understanding of the etiology and treatment of the disease has decreased the mortality rate. Certain features of spontaneous rupture merit individual mention, since early diagnosis and prompt institution of therapy are essential.

The majority of cases of spontaneous rupture of the esophagus are postemetic, usually in males (84 per cent),[18] and occur after a heavy meal or from repeated episodes of vomiting. A history of excessive alcoholic intake frequently is obtained. Usually there is no history of pre-existing esophageal disease, although a few cases have been reported with antecedent history of esophagitis, peptic ulcer, esophageal stricture or vomiting from neurologic causes.

Experimental observations show that the most important factor in producing rupture of a hollow viscus, such as the esophagus, is the rapidity of distention, rather than the total degree of pressure.[7] The majority of tears produced by sudden severe retching or vomiting are longitudinal and occur in the lower end of the esophagus on the left lateral wall. This has been shown by Mackler[13] to be the weakest part of the organ. Perforation may occur into the mediastinum, or the mediastinal pleura may be torn. Tear or rupture of the pleura causes a pneumothorax and empyema, usually on the left side.

The history of vomiting followed by severe substernal or epigastric pain should suggest the diagnosis. The pain usually is followed by a shock-like state, dyspnea and often cyanosis. The pain persists, but the vomiting usually stops after the perforation. Tachycardia and hypotension often are present, and the respirations may be labored. As the process progresses, subcutaneous emphysema may appear at the base of the neck and signs of a pneumothorax may be present, usually on the left.

DIFFERENTIAL DIAGNOSIS

The most common condition with which a spontaneous rupture of the esophagus can be confused is a perforated peptic ulcer. The rapidity of onset and severity of the pain also may frequently suggest an acute coronary occlusion. Other diseases which may mimic it are spontaneous pneumothorax, acute pancreatitis, dissecting aneurysm of the aorta, and pulmonary embolism. Spontaneous pneumothorax is the only other condition which may be present with subcutaneous emphysema and this is unusual. Consequently, the appearance of crepitation in the cervical region, occurring in at least 70 per cent of the reported cases of esophageal rupture, should direct attention to that diagnosis.[12]

Roentgenologic findings of mediastinal emphysema, with or without a pneumothorax, should confirm the diagnosis. The presence of gastric contents in aspirated pleural fluid also is pathognomonic. Lipiodol swallow also may confirm the diagnosis.

TREATMENT

Contamination of the mediastinal compartment by perforation of the esophagus may be compared in some ways to perforation of a peptic ulcer. In both situations, there have been recoveries in patients treated with gastric suction, massive chemotherapy and other supportive measures. Nevertheless, the best results will be obtained by surgical intervention. Ideally, the treatment is immediate closure of the esophageal tear, drainage of contaminated tissues and adequate nutrition of the patient.

The most important single factor in management of esophageal perforation is early recognition. Once the diagnosis has been reached, a decision must be made whether to close the perforation and drain the mediastinum or to institute drainage alone. As pointed out by Samson,[20] the main factors in consideration of therapy are the size and duration of the perforation and presence or absence of pre-existing esophageal disease.

Esophageal perforations without other esophageal disease, if seen within the first 24 hours, are best treated by closure of the tear and adequate drainage of the surrounding tissues. This should be by cervical mediastinotomy in the case of the cervical esophagus or by the transpleural approach in the case of the intrathoracic esophagus. Some surgeons may advocate an extrapleural approach through a posterior mediastinomy for treatment of an intrathoracic perforation. It is likely, however, that if an attempt is being made to close the perforation, exposure of the esophagus is less satisfactory, and closure more difficult, through a posterior mediastinotomy. In either case careful identification and closure of the perforation with interrupted silk sutures should be carried out.

Should the perforation be of more than 24 hours duration, or in the presence of advanced mediastinal suppuration, mediastinotomy with drainage is the procedure of choice. Samson, however, has advocated closure of a perforation of the cervical esophagus if recognized within 36 to 48 hours. Drainage of the retrovisceral space by cervical mediastinotomy usually is adequate in perforations of the upper one-third of the esophagus, since this space extends from the base of the skull to the bifurcation of the trachea. Perforations below this level are drained by posterior mediastinotomy. In all cases a levine tube should be inserted into the stomach. This may be passed under fluoroscopic control to avoid entering the mediastinum through the perforation. Massive doses of penicillin and streptomycin are started and the patient placed on nothing by mouth. Oxygen, digitalis, and other supportive measures may be carried out as indicated.

TECHNICAL CONSIDERATIONS

1. *Cervical Mediastinotomy:* This is done according to the technic of Pearse[16] and consists essentially of the following:

Under local anesthesia a low transverse cervical incision may be made on either side of the neck. Some prefer an incision parallel to the anterior border of the

sternocleidomastoid muscle. The sternocleidomastoid is retracted laterally, and the sternohyoid and sternothyroid muscles are split. The carotid sheath is retracted laterally, and the middle thyroid vein and inferior thyroid artery may be ligated and divided if necessary. The thyroid gland is rotated medially and by blunt dissection, the finger is passed back, lateral to the trachea and esophagus. Here the buccopharyngeal fascia may be felt and must be divided in order to enter the retrovisceral space. The esophagus is then freed up from the prevertebral fascia and several soft rubber tissue drains inserted in the retrovisceral space between the prevertebral fascia and esophagus. In early perforations there may be only a few air bubbles and some thin cloudy fluid detectable when the retrovisceral space is opened. In more advanced states when the space is under more pressure, decompression will be obvious as soon as the space is entered.

2. *Posterior Mediastinotomy:* This provides for extrapleural drainage of the mediastinum. The level of the mediastinal suppuration may be determined by roentgenogram. The surgical approach consists essentially of a vertical skin incision and resection of 6 to 8 centimeter segments of two or three ribs at the appropriate level with the corresponding transverse processes. Using blunt finger dissection, the pleura is reflected laterally until the mediastinal compartment is entered. The area of mediastinitis is drained with several soft rubber drains. The dressings should not be airtight because a large leak in the esophagus may continue to build up pressure in this compartment if the air is not allowed to escape. If, during mediastinotomy, the pleura is torn, a chest catheter should be inserted and attached to water-seal drainage.

Perforations which occur in the presence of an already diseased esophagus require certain modifications of the above treatment. Each case must be evaluated and a decision made whether to treat the perforation by closure, drainage, or definitive surgery of the underlying disease. In the case of acute chemical burns, drainage alone is indicated. Perforation by instrumentation in the presence of an esophageal carcinoma may occur. In this situation, if the lesion appears to be amenable to surgery, emergency thoracotomy and esophageal resection is justifiable. At the other extreme, if perforation of an obviously far advanced lesion occurs, supportive care alone is adequate.

AFTER CARE

Postoperatively it is necessary to suspend oral feeding until the tissues can heal. This time will vary with the size of the perforation and the patient's general condition. In the immediate postoperative period intravenous fluids should be given to maintain adequate fluid blance. Transfusions of blood usually are necessary. The gastric tube should be placed on continuous suction to keep the stomach decompressed. Antibiotics should be continued. Once peristalsis has resumed, nutrition may be maintained by feedings given through the gastric tube. Parenteral vitamins should be given. Tube feedings should be small in volume to avoid overloading the stomach, but may be given frequently. The patient should be kept in a sitting position to avoid reflux of the stomach contents into the esophagus. If for any reason, a gastric tube cannot be passed, gastrostomy or jejunostomy may be done for feeding purposes. If future esophageal resection with

gastroesophageal anastomosis is contemplated, jejunostomy is preferred. This will provide less difficulty in future mobilization of the stomach when resection is done.

The extent of healing of the esophagus may be tested by giving 20 to 30 milliliters of a methylene blue solution by mouth. The appearance of a blue stain on the mediastinotomy dressings signifies continued leakage of the perforation. In this period it is essential that adequate drainage of the mediastinum be maintained until closure of the perforation has occurred. Once the leak has closed, oral feedings may be resumed.

PROPHYLAXIS

Esophageal perforations may occur as a result of instrumentation despite all precautions. There are, however, certain measures to be stressed which may help to avoid this occurrence. The first is gentleness during esophagoscopy or dilatation. The instrument should never be forced. Muscle relaxation on the part of the patient is essential. This is best attained by doing esophagoscopies under general anesthesia whenever possible.

The presence of a sharp foreign body in the esophagus constitutes a potential perforation. This is particularly true with an object which is hooked or curved, such as a denture. If this cannot be dislodged and removed easily through the esophagoscope, we believe further attempts should be abondoned. The patient should be turned and a thoracotomy done on the appropriate side. The esophagus should be exposed, opened, and the foreign body removed. The esophageal opening may be closed in layers and the patient treated as a routine thoracotomy.

ILLUSTRATIVE CASES

Case 1. E. B., a 72 year old white man was admitted to the George Washington University Hospital on Oct. 27, 1953 because of dyspnea, anorexia and fatigability. He also gave a history of epigastric discomfort, and had noted tarry stools for several weeks prior to admission.

Physical examination on admission showed a well developed, moderately well nourished, elderly white man. His lungs were clear. A Grade III apical systolic murmer was present. The blood pressure was 154/74. The liver was palpable 2 fingerbreadths below the costal margin. There was evidence of some fluid in the abdomen and vibratory sense in the lower extremities was diminished.

Laboratory studies: The hemoglobin was 5.7 grams per cent; the hematocrit 20; a stool test for blood showed 4 plus guaiac; the urine was normal.

Course in the hospital. After admission the patient was given several transfusions for his anemia. A gastrointestinal roentgenologic study showed pyloric obstruction with marked dilatation of the stomach. There was 90 per cent six hour retention of barium in the stomach. On the morning of the fifth hospital day the patient felt nauseated and vomited a large quantity of fluid. Almost immediately he complained of upper abdominal and substernal pain. When seen by a physician he was in respiratory distress. The blood pressure was 80/60, pulse 110, and respirations 30. It was noted that there was crepitation of the tissues of the neck. Roentgenograms showed mediastinal emphysema and a hydropneumothorax on the left. He was treated with oxygen, vasopressors and gastric suction with little improvement. The next morning he was seen by the thoracic surgeons. At that time a presumptive diagnosis of esophageal rupture was made. He was given lipiodol by mouth and this appeared in the left pleural cavity as shown by roentgenogram.

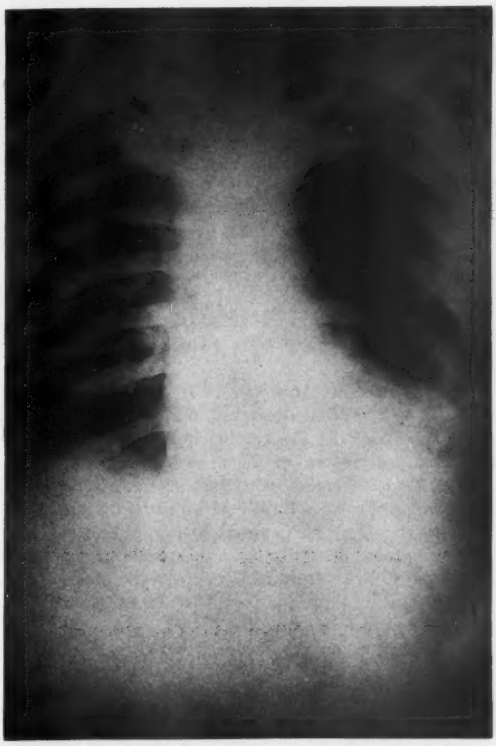

FIG. 4. Case 1, 16 days after esophageal rupture, showing clearing of mediastinal emphysema and re-expansion of left lung. Note presence of pleural reaction and some effusion on right.

At this time the patient was in a precarious condition and it was thought that he was not a satisfactory risk for thoracotomy. Intercostal tubes were inserted in the left chest, one in the third interspace anteriorly and another in the eighth interspace posteriorly. Fifteen hundred milliliters of cloudy, straw colored fluid drained from the chest within the next few hours. Supportive treatment in the form of digitalis, antibiotics and intravenous fluids was given. A plexitron tube was passed into the stomach.

During the next eight days the patient's general condition improved. He subsequently developed a right pleural effusion which was aspirated on three occasions (fig. 4). On the nineteenth hospital day a gastroenterostomy and jejunostomy were done under local anesthesia for feeding purposes. At that time a large tumor mass involving the pylorus, duodenum, pancreas and mesentery was noted. A biopsy section of this mass was reported as anaplastic adenocarcinoma.

The patient gradually went downhill and died on the twenty-third hospital day.

Autopsy showed a tear 4 cm. in length of the left anterolateral wall of the esophagus, just above the cardia. There was communication with the left pleural cavity. No tumor was seen in the esophagus. There was a large adenocarcinoma of the stomach with extensive involvement of other abdominal organs.

Comment: This case represents a postemetic rupture of the esophagus, secondary to a pyloric obstruction from carcinoma. The site of rupture and the clinical manifestations

were classical. Had the condition been recognized earlier, closure of the esophageal tear might have been feasible. The patient did respond satisfactorily to decompression and drainage of the left pleural cavity, however, and might have survived had it not been for the advanced malignancy. It is worthy of note that decompression of the mediastinum occurred by rupture into the left pleural cavity. Had this not taken place, death might have occurred within the first 24 hours from mediastinal emphysema and tamponade.

Case 2. M. S., a 36 year old white woman was admitted to the George Washington University Hospital on Sept. 3, 1949. Eight days previously she had fainted, fell and swallowed a dental bridge. She was taken to another hospital where esophagoscopy was done. Apparently the bridge had been pushed into the stomach, but an esophageal tear had occurred, since subcutaneous emphysema was noted in the neck shortly thereafter. She was given nothing by mouth and given penicillin and streptomycin. Her temperature reached 102.4 Farenheit on her third hospital day and remained in the region of 101° F., subsequently. Attempts to aspirate pus from her chest were unsuccessful. She was transferred to this hospital on her eighth day.

Physical examination: The temperature was 100.4 F., pulse 84, respiration 34, and blood pressure 149/86. She was a well developed, slightly obese white woman, pale and breathing heavily. She obviously was quite ill. The trachea was deviated slightly to the right and subcutaneous emphysema was present in the neck and upper thorax. Moist rales were heard in both lung fields. Tactile fremitus and breath sounds were diminished on the right. Roentgenograms of the chest showed a right hydro-pneumothorax. Abdominal roentgenograms showed the denture to be present in the right lower quadrant.

Course: On the day of admission, under local anesthesia, a segment of the ninth rib on the right was resected and water-seal drainage of the right pleural cavity was instituted. About 2000 milliliters of thin fluid drained from the pleural cavity. On the following day the right lung was almost completely expanded.

She continued to run a septic course, however, and chest roentgenograms showed further mediastinal widening. On her fourth hospital day a posterior mediastinotomy was done under general anesthesia, resecting portions of the right eighth and ninth ribs. A moderate quantity of purulent material was encountered. On her seventh hospital day, her temperature rose to 104 F. and marked widening of the superior mediastinum was noted on the roentgenogram. A cervical mediastinotomy was done under local anesthesia. Following this the patient drained profuse quantities of pus from the cervical wound. She made a gradual recovery and became afebrile. The denture remained in the region of the ileocecal valve. On the twenty-sixth hospital day a laparotomy was done and the denture was manipulated into the colon where it was quickly passed. She was discharged on Oct. 8, 1949.

Comment: Considerable discomfort and hospitalization might have been avoided had a thoracotomy been done on this patient in the initial phases of her difficulty. If the denture did not move easily during esophagoscopy, transthoracic removal would have been indicated. Likewise, the esophageal tear, recognized within a few hours of its occurrence, would have been best treated by immediate thoracotomy and suture of the tear. This case also is a good example of the difficulties encountered in treatment of an acute suppurative mediastinitis.

SUMMARY AND CONCLUSIONS

Esophageal perforations constitute a surgical emergency.

Early diagnosis of a perforation is paramount in the successful treatment of this situation.

The diagnostic points and outline of treatment have been discussed.

Drainage of the mediastinum is essential in all cases.

Closure of the perforation and drainge of the mediastinum is advocated if the

condition is recognized within 24-hours, unless contraindicated by pre-existing disease.

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RETICULUM CELL SARCOMA: A CASE POSSIBLY ORIGINATING IN REGIONAL ENTERITIS

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Since 1932, when Crohn, Ginzburg, and Oppenheimer[4] established regional enteritis is a distinct clinical and pathologic entity, many papers on this subject have appeared in the literature. Only a few writers have mentioned the association of regional enteritis with malignancy of the small bowel. Most of them have believed that there is no such relationship.[3, 8] In a review of the English literature since 1932, we could find only one author who believed that there was a possible correlation between sarcoma of the small intestine and regional enteritis.[7] To our knowledge, no case of malignant degeneration of regional enteritis has been reported in the English literature.

Because of the possibility that Crohn's disease may undergo malignant degeneration, we report the following case:

CASE REPORT

Case History: A 72 year old government office worker was admitted to the George Washington University Hospital on Oct. 7, 1954, because of abdominal distention and pain.

Sixteen years before admission he had been struck in the abdomen by the fender of an automobile. Immediately thereafter he developed distention, cramping abdominal pain, constipation, and intermittent diarrhea. Six weeks later he noticed visible intestinal movements and heard loud bowel sounds, especially after eating. The distention, abdominal pain, and diarrhea gradually became more severe, and he lost 30 pounds in weight. Some relief was obtained by a low residue diet and enemas. Two gastrointestinal roentgenograms four months after the accident were reported to reveal marked dilatation of the ileum.

Fourteen months after injury, he was hospitalized again because of similar symptoms. Physical examination again showed abdominal distention, visible and audible peristalsis. Routine laboratory tests, including stool examinations, were within normal limits. Sigmoidoscopy showed no abnormality. At laporotomy a thickened ileum dilated to four times its normal size was encountered. Sixty cm. proximal to the cecum, a 15 cm. segment of thickened stenotic ileum was resected. On opening the specimen, ulceration was present, and the lumen was only large enough to admit a lead pencil. The post-operative course was prolonged, but uneventful, and the patient was discharged in good condition.

The pathologic diagnosis of the specimen was nonspecific inflammatory lesion of the ileum compatible with regional enteritis (fig. 1.).

During the following 15 years, his health gradually deteriorated. His weight dropped from 180 to 142 pounds. The diarrhea steadily became more severe. There often were as many as 10 to 20 loose stools in one day. He had frequent bouts of cramping abdominal pain and grew more weak and fatigued on slight effort. During frequent examinations his abdomen was usually distended, tympanitic, and filled with visible bowel loops. Repeated small bowel roentgenograms were reported as showing dilated loops of ileum and partial intestinal obstruction.

During the nine months prior to entry into the George Washington University Hospital, he began to complain of dull right flank and right upper quadrant abdominal pain in addition to the abdominal cramping which also was growing more severe.

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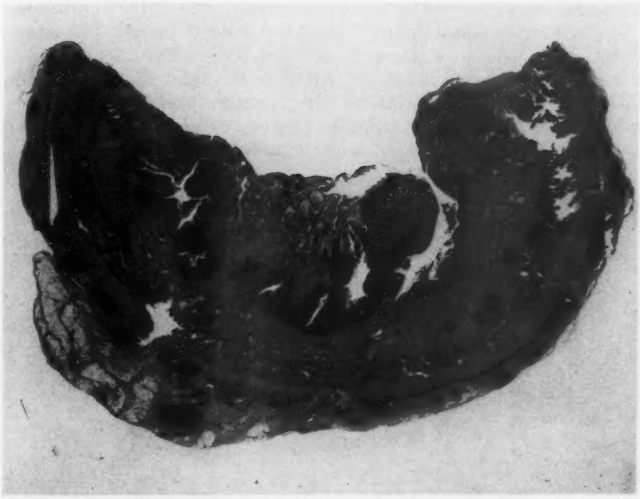

FIG. 1. Cross section of ileum at the thickened, stenotic, ulcerated site. Pathologic diagnosis: regional enteritis.

One month before entry, he passed dark, bloody stools on two occasions. He became more distended, had occasional episodes of nausea and vomiting, and grew more confused mentally. Ankle edema developed and a tender mass was palpated in the right lower quadrant, which increased rapidly in size during the month.

Two weeks prior to entry, a small bowel roentgenologic study was reported as showing two narrowed segments of ileum with consecutive dilatation of the small bowel loops.

Physical Examination: He was a tall, thin, cachectic, pale, white man. The temperature was 36.4, the pulse 80, and the respirations 20. The palms were erythematous, the eyes sunken, and the musculature atrophic. The abdomen was distended and of irregular contour. There was an old right rectus scar. Amid huge dilated bowel loops, movable, hard, tender masses could be palpated in the right lower quadrant. The entire abdomen was tympanitic, and loud, gurgling bowel sounds could be heard.

Laboratory Studies: The urine contained three plus protein. The hematocrit was 35 mm. and the white cell count 9,250 per cu. mm. with 76 per cent neutrophiles, 22 lymphocytes, and 2 monocytes. The total protein was 4.64 Gm. per cent, the globulin 1.74 Gm. per cent, the albumin 2.90 Gm. per cent, the blood urea nitrogen 17 mg. per cent, cholesterol 287 mg. per cent, cholesterol esters 63 per cent, bromsulphalein retention 10 per cent and prothrombin activity 64 per cent. Fasting blood sugar was 119 mg. per cent with a very flat curve, probably indicating poor intestinal absorption. Serum chlorides, sodium, and potassium were within normal limits.

Roentgenologic Examination: A film of the abdomen showed partial small bowel obstruction.

Hospital Course: During the first 10 days in the hospital, his condition was much as it had been before entry. On the eleventh day, he began to pass dark blood per rectum and became semistuporous. The blood urea nitrogen rose to 26 mg. per cent. Three blood transfusions totalling 1500 cc. were given. A small quantity of peritoneal fluid was aspirated and found to contain malignant, non epithelial tumor cells, probably indicating reticulum cell sarcoma. Culture of the peritoneal fluid showed E. coli. His condition deteriorated rapidly and on the sixteenth day he died.

Autopsy: A 9 by 11 cm. tumor mass was found encircling the ileum 34.5 cm. proximal to the caecum, and 37 cm. proximal to this was another tumor measuring 11 by 12 cm. At both

levels the lumen of small bowel was almost completely obstructed and the mucosa was ulcerated. The ascending colon contained two small nodular tumors. The mesenteric and retroperitoneal lymph nodes were enlarged, confluent, and involved in tumor tissue. Tumor also was present in the internal mammary, left supraclavicular, periaortic, and peribronchial lymph nodes. The culdesac was matted with tumor and scattered nodules were present about the peritoneum. Several small tumor nodules were found in the lungs and liver. The microscopic diagnosis was reticulum cell sarcoma. (See figures 2 and 3).

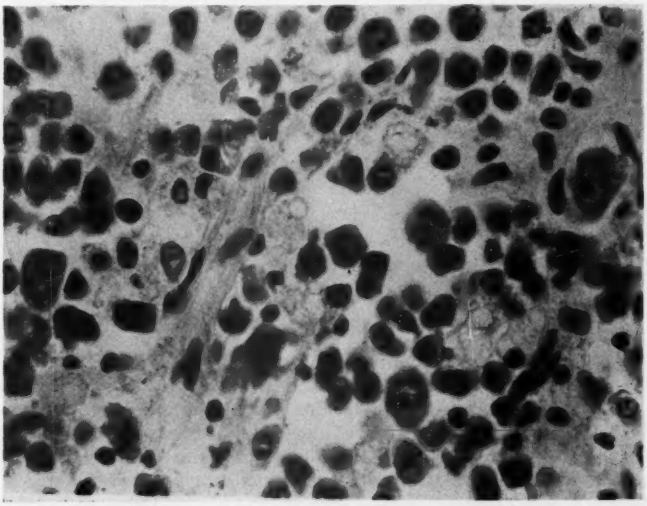

FIG. 2. A high power view of the reticulum cell sarcoma in the small bowel wall.

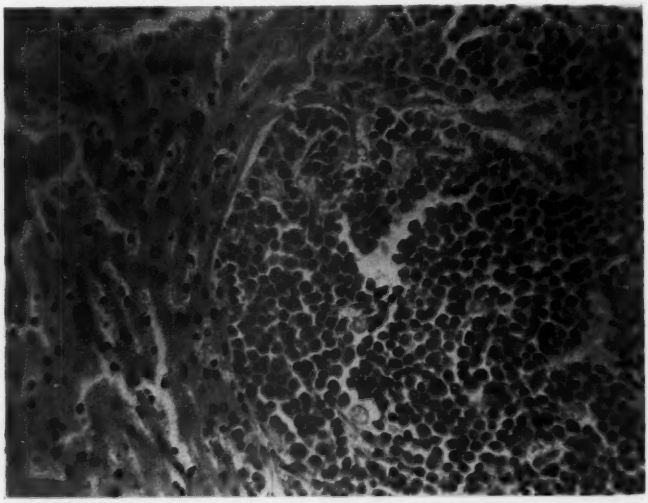

FIG. 3. A high power view of a metastases of the reticulum cell sarcoma to the liver.

DISCUSSION

Because of the previous pathologic findings and the course of the disease, we believed at first that this was an advanced case of regional enteritis which had followed blunt abdominal trauma.[1, 2, 5, 6] However, with the enlarging abdominal masses and the rapid deterioration of the patient's condition, we began to suspect the possibility of malignancy. Our suspicions were confirmed when the peritoneal fluid cell block contained malignant nonepithelial tumor cells.

More interesting were the postmortem findings. The same area in which a thickened, chronically inflamed segment of bowel had been resected 14 years earlier now was diffusely involved in reticulum cell sarcoma. We suggest the possibility that this case of regional enteritis did actually undergo sarcomatous degeneration.

SUMMARY

A case of regional enteritis following blunt abdominal trauma believed to have degenerated into reticulum cell sarcoma is presented.

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PSEUDOCYSTS OF THE PANCREAS

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Pseudocysts of the pancreas are infrequently encountered on the average surgical service, so that individual experience with them is limited.

The complicated pathologic findings of this disorder often make their surgical management most difficult. In no way can their treatment be standardized. Our experience has lead us to favor an external drainage procedure initially, followed, when necessary, by excision of the involved portion of the pancreas or an internal drainage procedure.

The differentiation of pseudocysts of the pancreas from other types of pancreatic cysts often depends upon the operative findings. Occasionally the correct diagnosis can be suspsected on the basis of the clinical study. Frequently not only is it impossible to determine the type of cyst preoperatively, but it may be impossible to differentiate a pancreatic cyst from other upper abdominal lesions.

CLASSIFICATION

Pancreatic cysts have been classified in many ways, but the classification based upon etiologic consideration seems most useful:

- Developmental
  - Fibrocystic disease
  - Retention cysts
- Inflammatory
  - Resulting from pancreatitis
- Traumatic
  - Rupture of pancreatic ducts
- Neoplastic
  - Cystadenoma
  - Cystadenocarcinoma
  - Dermoid
- Parasitic

According to this classification pseudocysts belong to the second and third groups, i.e., the inflammatory and the traumatic cysts.

ETIOLOGY

Pseudocysts develop when a pancreatic duct is ruptured and pancreatic secretions are allowed to escape into the surrounding tissue. Ductal rupture may result either from trauma or attacks of acute pancreatitis. The development of a pseudocyst may be delayed for a considerable time after blunt abdominal trauma. It may develop suddenly in acute pancreatitis when the pancreatic capsule ruptures

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allowing fluid to accumulate in the lesser peritoneal sac. With the escape of pancreatic secretions there is a surrounding tissue reaction with resulting encapsulization of the fluid. The capsule of the cyst thus is a false one, being composed of the surrounding organs and mesenteries.

The cysts may be single or multiple, and frequently are multiloculated. Their contents may vary from clear fluid to saponified necrotic debris.

When the enzymes escape into the tissue spaces they may be activated and digest tissue. At times nearly the whole pancreas may slough. Secondary hemorrhage may occur into the cyst. When a cyst is found to contain clear fluid a communication between the cyst and a pancreatic duct usually can be demonstrated. This represents a late stage, in which necrosis and inflammation have subsided and the pancreatic secretions in the cyst no longer are activated. This type of cyst must be carefully differentiated from the cystadenoma which produces large quantites of clear fluid in a cyst even though the tumor may be small. In addition to the recognition of a true capsule in the latter condition the amylase content of the fluid may be helpful in the differentiation. Since all pseudocysts result from duct rupture the fluid in the cyst will contain amylase. With a cystadenoma the fluid in the cyst is secreted by the tumor and will not contain enzymes.

A pancreatic pseudocyst may regress spontaneously. They may drain via an open pancreatic duct. This factor is the rationale for treating a pseudocyst by sphincterotomy in an attempt to promote proper drainage of pancreatic secretions through the ampulla of Vater. When there is no obstruction to this route of outflow, pancreatic secretions will not accumulate in the cyst and the cyst will tend to drain through the pancreatic ductal system. At times pseudocysts may drain spontaneously into the adjacent viscera. They drain most frequently into the transverse colon and the stomach. Secondary empyema on the left side is not unusual. There have been patients in whom the cyst drained directly into the pleura and then escaped by being evacuated through a bronchopleural fistula.

Usually spontaneous regression of the cyst does not occur and the upper abdominal mass continues to enlarge. There may be compression of surrounding viscera. The symptoms produced are varied. A cyst may be detected as a painless, enlarging abdominal mass with no associated symptoms. A history of previous abdominal trauma frequently can be elicited in such instances. Most of the cysts that result from acute pancreatitis are associated with severe symptoms consisting of upper abdominal pain which may radiate through to the back, digestive disorders, nutritional deficiencies, fever and abdominal distention. Occasionally a large cyst may rupture and leak into the peritoneal cavity leading to signs of diffuse peritonitis.

DIAGNOSIS

The diagnosis of a pseudocyst of the pancreas is suspected when an upper abdominal mass develops following abdominal trauma or develops in a patient with acute recurrent pancreatitis.

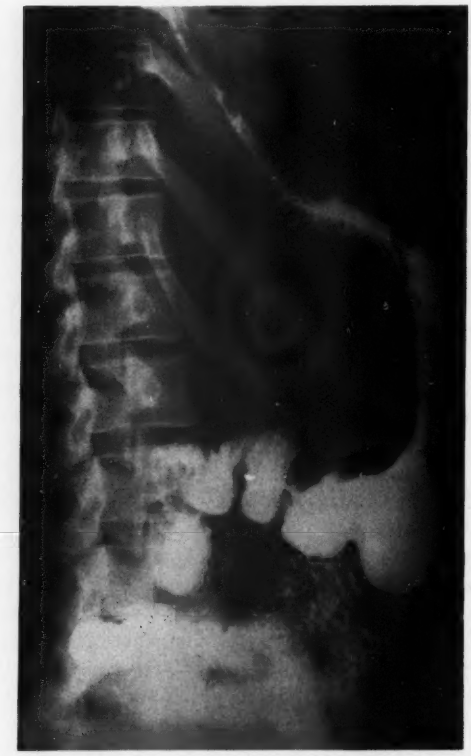

FIG. 1. Note the large extrinsic pressure defect of the pseudocyst displacing the stomach to the left and anteriorly.

The diagnosis of cases of patients presenting an asymptomatic mass depends upon excluding other upper abdominal lesions. In addition to upper and lower gastrointestinal tract barium studies, pyelography, retroperitoneal air insufflation studies and aortograms may be helpful. In the presence of a large cyst, the stomach will be displaced anteriorly (fig. 1). Widening of the duodenal loop occurs when the cyst is located in the head of the pancreas (fig. 2). The transverse colon and the splenic flexure may be displaced inferiorly. Pyelograms help to exclude renal lesions. In patients that present with an upper abdominal mass and the signs of an acute abdominal condition the serum amylase test and an amylase determination on the peritoneal fluid are most helpful in establishing the diagnosis. A specimen of peritoneal fluid can be obtained by needle tap of the abdomen.

The operative diagnosis depends upon the differentiation between true pancreatic cysts and pseudocysts. This differentiation is made by close inspection, biopsies, and amylase determination of the fluid content of the cyst. The pseudocyst has a fibro-inflammatory capsule in which the surrounding viscera and

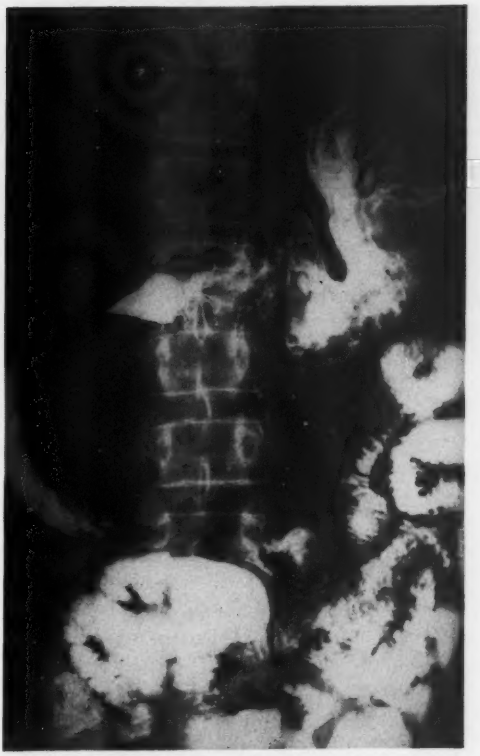

FIG. 2. Note the widening of the duodenal loop caused by a pseudocyst in the head o the pancreas. Also note the calcifications in the head of the pancreas.

mesenteries may be included. This usually prevents a complete excision of the cyst. In the neoplastic cysts, however, there is a true capsule which frequently is not adherent to the surrounding viscera. In such cases complete removal is feasible. After determining a cyst is not removable, the cyst should be opened, and its interior inspected. Careful note should be made of any papillary projections into it, and if any are present a biopsy and frozen section study should be made. This study is essential to exclude cystadenoma and cystadenocarcinoma. Biopsy and frozen section study of the cyst wall is done to verify its inflammatory nature and thus distinguish it from the true cyst. The fluid of the cyst may be analyzed for amylase content. The presence of pancreatic enzymes indicates a pseudocyst.

TREATMENT

Pancreatic cysts require surgical therapy. True cysts should be widely excised. Pseudocysts may be treated by (1) simple drainage; (2) marsupialization; (3) excision; (4) internal drainage; (5) resection; and (6) sphincterotomy.

Simple drainage is the easiest method of external drainage, and is advised in all poor-risk patients or in patients in whom other forms of therapy are hazardous. Marsupialization is preferred for external drainage whenever it is possible to suture the cyst wall to the skin.

Internal drainage is obtained by anastomosis of the cyst to the gastrointestinal tract. Direct anastomosis between the cyst and the stomach, duodenum or jejunum will allow intestinal contents to accumulate in the cyst cavity. The most satisfactory type of internal drainage is a Roux en Y cystojejunostomy, with a long defunctionated limb so that intestinal contents can not enter the cyst.

Excision or resection is the method of choice whenever feasible. Unfortunately this seldom is possible in pseudocysts because of the danger of injury to adjacent viscera in the false capsule.

CLINICAL MATERIAL

During the period of 1950 to 1954, 9 cases of pseudocysts of the pancreas have been treated at the District of Columbia General Hospital. During these years no case of any other type of pancreatic cyst has been encountered.

Eight of these 9 cases occurred in patients who were addicted to alcohol and who had had repeated attacks of acute pancreatitis. In 1 patient a cyst developed following laceration of the pancreas by blunt abdominal trauma.

In 6 patients a smooth mass was palpable in the epigastrium or left upper abdominal quadrant. In the remaining 3 patients a mass was not palpable. One of these patients was operated upon for diffuse peritonitis, which was thought to be due to a perforated ulcer. A ruptured pseudocyst was found. Another of these patients was explored for obstructive jaundice and a pseudocyst of the head of the pancreas was found compressing the common duct. In the other patient, a cyst was demonstrated only by gastrointestinal roentgenogram.

The serum amylase was elevated preoperatively in 8 of the patients. Upper gastrointestinal roentgenograms confirmed a diagnosis of a lesser peritoneal space-taking mass in 7 patients. In all 9 patients the cyst fluid was found to contain amylase. Pathologic examination of the cyst wall in each patient showed chronic inflammatory tissue. In 3 patients sections of regional lymph nodes were made to exclude malignancy.

Six patients were treated by simple external drainage. In 2 patients a Roux en Y cystojejunostomy was made. In 1 patient excision of the pseudocyst was possible.

The patients treated with external drainage recovered rapidly. The fistulous tract that developed drained for approximately three to six weeks, then spontaneously closed in all instances. None has developed a recurrent cyst or a recurrent fistula tract. The 2 patients treated by internal drainage have done well. In 1 of these patients a cholecystectomy was done for gallstones three months later. During the operation the cystenterostomy was explored and found to be closed with a complete disappearance of the cyst.

All patients have been followed to date, with follow-up periods ranging from

one to five years. Six patients now are well with no residual. Two patients now have moderate pancreatic insufficiency manifested by failure to gain weight, intermittent foul bulky stools, and one has diabetes. The only death in the group occurred in the patient treated by excision of the cyst. This patient died on the twentieth postoperative day following the development of a duodenal fistula. The cyst in this patient was located in the head of the pancreas and its removal apparently jeopardized the blood supply of the duodenum.

DISCUSSION

Pseudocysts of the pancreas may be handled surgically in numerous ways. We favor external drainage as the initial procedure and marsupialization when possible. The drainage tract is kept open until it spontaneously closes from within outward. Closure of the tract before the cyst has healed must be prevented or a cyst is apt to redevelop. In view of our satisfactory experience with external drainage without recurrence or persistent draining fistula, we favor this treatment to an internal drainage procedure. Although internal drainage is a satisfactory operation apparently it is not necessary when most of the pancreatic cysts will close with simple external drainage. It was extremely interesting to note the complete closure of the cyst in the patient we had the opportunity to re-explore after an internal drainage procedure. The cystenterostomy handled the initial problem of drainage of the cyst satisfactorily, but was not necessary for a permanent drainage since the cyst closed and apparently the ductal communication sealed.

If the external drainage tract remains open after three months, radiopaque material should be injected into it to determine any residual cavity or the presence of a ductal communication. If either of these is present, further surgery is indicated. During this period of time a patient may be gotten into optimum condition for elective surgery. At the second operation we favor excision of the fistulous tract with the involved portion of the pancreas if the body or tail of the pancreas is involved. If the head of the pancreas is involved, a Roux en Y cystojejunostomy is preferred to excision because of the danger of injury to the duodenum or its blood supply. In either instance the tract is excised. We do not advocate implanting the fistulous tract into the bowel, for it is apt to stenose and then a cyst will redevelop. Whenever a second operation is required either the cyst should be excised or a permanent form of internal drainage established. In these patients a large ductal communication exists and unless it is closed or allowed to drain properly further difficulty is to be anticipated.

SUMMARY AND CONCLUSIONS

Pseudocysts of the pancreas result from either abdominal trauma or as a complication of pancreatitis. In either instance a pancreatic duct is ruptured and pancreatic secretions escape and a false capsule develops.

The diagnosis of a pseudocyst can be anticipated in a patient with an upper abdominal mass with a previous history of abdominal trauma or acute pancreatitis. Roentgenologic studies may show displacement of the stomach or duodenum.

The correct diagnosis depends upon the operative findings which must differentiate a pseudocyst from a true pancreatic cyst or pancreatic neoplasm, since the treatment is different.

The methods of treatment of pseudocysts have been outlined. Preference for initially creating external drainage has been advocated. If the fistulous tract fails to close or a cyst redevelops the involved portion of the pancreas should be resected or internal drainage by a Roux en Y cystojejunostomy should be established.

Experiences with 9 cases of pseudocysts of the pancreas have been briefly presented.

ADENOCARCINOMA OF THE ESOPHAGUS ARISING IN ABERRANT GASTRIC MUCOSA

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The occurrence of aberrant gastric mucosa in the esophagus is unusual and was first described by Schmidt[5] in 1805. Since that time there have been sporadic case reports in the literature concerning this anomaly.

Rector and Connerly[4] found in 1000 consecutive autopsies in children, 80 cases of heterotopic gastric mucosa in the esophagus. The sections were taken at random and at various levels. It is interesting that only in 26 of the 80 cases were parietal cells found in the mucosa. They also stated that no gastric mucosa was identified below the muscularis mucosa. Feldman[3] reported a case of adenocarcinoma arising in a pedunculated polyp in the upper esophagus. Carrie[2] described a similar case of adenocarcinoma which arose from aberrant gastric epithelium in the upper portion of the esophagus. Bosher and Taylor[1] recently described a case of heterotrophic gastric mucosa in the esophagus with ulceration and stricture formation. The patient was successfully treated by esophagectomy and supra-aortic esophagogastrostomy.

The clinical significance of aberrant gastric mucosa in the esophagus in relation to stricture formation and malignant transition has not received sufficient attention.

The following case report concerns a patient with adenocarcinoma of the esophagus arising in the middle third of the organ. Esophagectomy and supra-aortic esophagogastrectomy, employing a right-sided approach, was carried out. Ten days after the operation there was disruption of the anastomotic line and the patient died shortly thereafter. Microscopic examination of the anastomotic site revealed the presence of carcinoma which was not grossly visible. It further revealed that the esophagus was completely replaced by gastric mucosa, from cardia to just a few centimeters below the pharynx.

CASE REPORT

A 34 year old white man was admitted to the George Washington University Hospital with a complaint of dysphagia of two months' duration. The patient had particular difficulty in swallowing solid foods and hot liquids. There was no anorexia, weight loss, chest pain, hematemesis, vomiting or regurgitation.

The past history was particularly revealing, since this patient had been treated for duodenal ulcer at intervals during the past two years. Moreover, he had been discharged from the Army with the diagnosis of psychoneurosis, anxiety state, because he constantly complained of digestive difficulties and some pain. However, examination of the gastrointestinal tract with barium did not show a lesion.

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FIG. 1. Roentgenogram of barium examination showing the constricting midesophageal lesion.

At the time of his admission, examination of the esophagus with barium swallow showed a middle-third constricting lesion (fig. 1). Examination through the esophagoscope showed a fungating lesion in the middle third of the organ. The original biopsy was reported as highly undifferentiated epidermoid carcinoma.

A right thoracotomy was performed and the lesion was located in the middle thoracic esophagus. The organ was mobilized from the level above the azygos vein to the diaphragm without difficulty. The stomach was freed through a left rectus incision, passed into the thorax and an esophagectomy and esophagogastrostomy were performed. The anastomosis was well above the level of the arch of the aorta in the apex of the right thorax. The esophagus appeared grossly involved from about 5 cm. below the level of the resection to the gastric cardia.

The postoperative course was uneventful until the tenth postoperative day. At that time there was temperature elevation, dyspnea, cyanosis, hypotension and marked tachycardia.

On roentgenologic examination a right hydropneumothorax was evident. A chest catheter attached to a water seal drainage was placed in the thorax. The drainage from the catheter had the appearance of gastric contents. The patient died within a few hours.

Gross Appearance of Surgical Specimen: The specimen consisted of a segment of esophagus measuring 11.5 cm. Starting at the upper end and extending distally for a distance of 7 cm., there was an annular, constricting neoplasm. The mucosa about the neoplasm was thin, fixed and superficially ulcerated in places. The wall was firm and measured as much

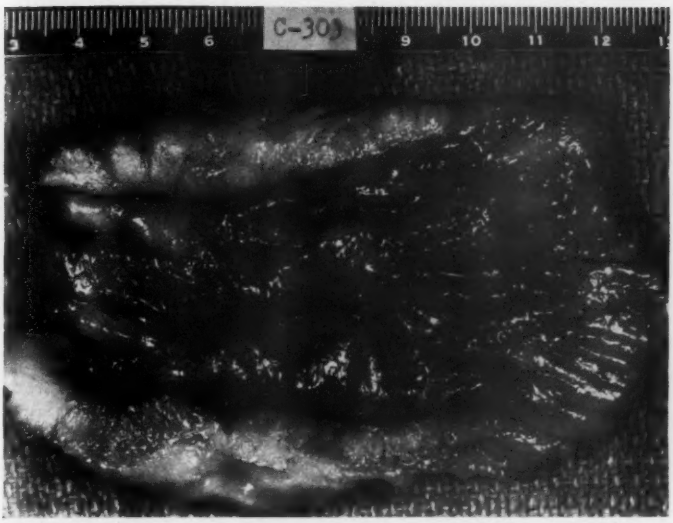

FIG. 2. The gross specimen showing the thick wall, relatively smooth lining, and cylindrical character of the neoplasm.

as 1.2 cm. in thickness. Near the center the muscularis was completely replaced by gray tumor tissue for a distance of about 1.5 cm. Above and below this zone, strands of similar tissue could be seen extending in a radial fashion between the muscle bundles and completely through the wall (fig. 2). Ten lymph nodes were found in the periesophageal tissues.

Microscopic Description: In the central portion, the mucosa was entirely lacking, and the lining was formed by tumor cells of an undifferentiated character supported by a moderate amount of fibrous stroma. At either end the esophageal mucosa consisted of branched tubular glands lined by tall columnar cells of both mucous and Paneth types and resembled gastric mucosa (fig. 2). Infiltrating between these glands and into the submucosa, were anaplastic tumor cells showing no significant structural characteristics (fig. 3). In the more central portions the carcinoma cells infiltrated completely through the muscularis. In the depths of the tumor, however, there was considerable differentiation, and well-formed glandular structures were evident (fig. 4). Four of the 10 regional lymph nodes contained metastases.

Postmortem Examination—Gross Description: There was about 200 cc. of bloody fluid in the left pleural cavity and 400 cc. of similar fluid in the right. The parietal and visceral pleura on the right were partially adherent by fibrinous adhesions. The left lung weighed 500 Gm. and was atelectatic. The right lung weighed 700 Gm. and also was atatic.telec Several small branches of the pulmonary artery were occluded by thrombi.

The greater part of the stomach was in the thorax and anastomosed to the proximal esophagus. A rupture 2 cm. long was present in this anastomosis, and the surrounding tissues and mediastinum were congested, edematous and indurated. The proximal esophagus was lined by pale gray, wrinkled mucosa. The character of this mucosa changed abruptly about 2.5 cm. above the anastomotic site. From this point to the gastric fundus, the lining was soft, pink-gray and identical in appearance with the mucosa of the cardia of the stomach.

Microscopic Description: The upper portion of the esophagus was lined by stratified squamous epithelium (fig. 3). About 2½ cm. from the proximal end of the esophagus the

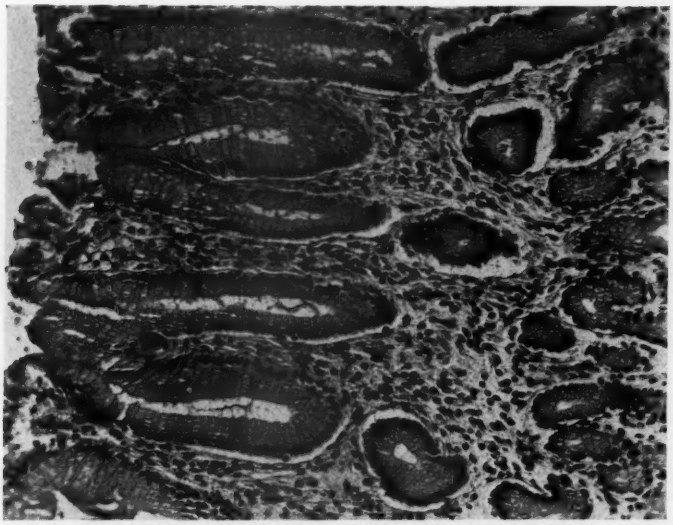

FIG. 3. Esophageal lining beyond the tumor edge showing its close resemblance to gastric mucosa.

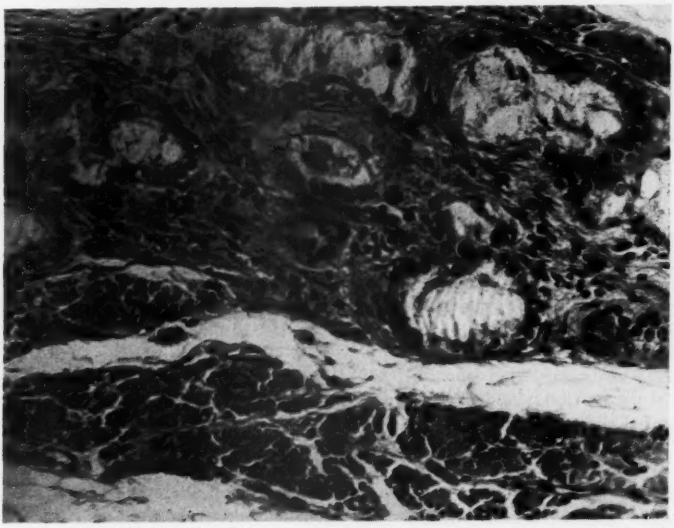

FIG. 4. A better differentiated focus of the carcinoma among the muscle bundles of the esophageal wall.

mucosa changed abruptly to the glandular type similar to that in the gastric cardia. A few clusters of residual tumor cells were found in the region of the anastomosis.

SUMMARY

This unusual case is reported to illustrate the significance of aberrant gastric mucosa in the esophagus, which underwent malignant transformation.

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FIBROSARCOMA ARISING IN A "JUVENILE" NASOPHARYNGEAL ANGIOFIBROMA FOLLOWING EXTENSIVE RADIATION THERAPY

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Recent papers have emphasized the clinical[2, 7] and pathologic[8] features of the juvenile nasopharyngeal angiofibroma. It is a relatively rare, histologically benign, suggestively age and sex-linked tumor, most frequently found in adolescent boys. These lesions have been treated by surgical morcellation or irradiation or combinations of these modalities.

The present case is of particular interest because it first became manifest six years ago in a now 54 year old man, who ultimately was treated by employing a different type of surgical approach than used heretofore for this tumor, and whose lesion showed areas of fibrosarcoma in a nasopharyngeal angiofibroma, approximately 10 months after extensive irradiation (9000 roentgens tumor dose).

CASE REPORT

The patient is a 54 year old white man whose difficulty began six years previously when he developed headaches and intermittent obstruction of the right nasal cavity. Past history indicated a nasal fracture at age 10 years with no residua. He had had normal maturation, was married and was the father of two children. The positive findings in 1949 were limited to the nasopharynx and nasal cavity. On anterior rhinoscopy, the right nares was completely, and the left nares partially, occluded, although no tumor mass could be seen. On mirror examination of the posterior nasopharynx, a rounded, smooth mass protruded from the right nares and extended along the right lateral and superior walls of the nasopharynx. The remainder of the physical examination, including the genitalia was normal. Secondary sex characteristics were well developed. Roentgenograms of the skull showed a soft tissue mass in the right nasal cavity and nasopharynx. A biopsy was taken and reported as "angiofibroma, juvenile type." On Feb. 15, 1949 after ligation of both external carotid arteries, the tumor was approached through the right maxillary antrum using a Weber-Ferguson type of incision through the upper lip and along the right naso-labial fold. The anterior wall of the right antrum, naso-antral wall with most of the ethmoidal sinuses, and the right nasal turbinates were removed.

The tumor then could be visualized in the superior portion of the right posterior nares. All the tumor visualized was removed without too much difficulty and with no untoward bleeding. In retrospect, the removal probably was incomplete. His postoperative course was uneventful, and symptoms were relieved for over a year.

The excised tumor measured 2.5 by 1.7 by 1.2 cm. and was classified as a fibroma (fig. 1).

His first recurrence was marked by right nasal obstruction, this time accompanied by bouts of epistaxis. A tumor again was visualized in the right nasopharynx with some bulging along the right lateral pharyngeal wall. On palpation, it was found to involve the floor of the nasal cavity, soft palate and right lateral pharyngeal wall.

On Jan. 2, 1952, the nasopharynx was re-explored by Dr. John Blady at Temple University Hospital through a flap incision on the right side of the soft and hard palate. The

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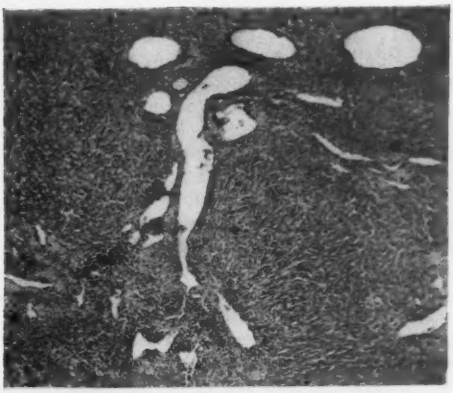

FIG. 1. Original lesion (1949) illustrating the two features of the tumor. An angiomatous framework consisting of simple endothelial lined channels and a connective tissue stroma with fine and coarse collagen fibrils, interspersed with connective tissue cells. ×50.

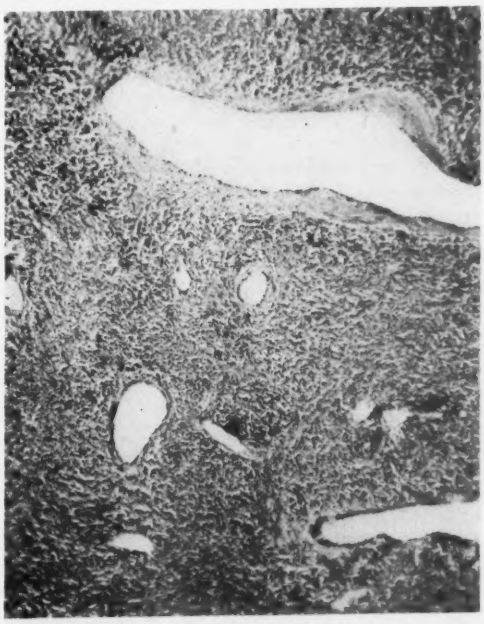

FIG. 2. Incompletely resected tumor (1952) prior to irradiation; no marked differences from preceding figure. ×100.

tumor mass was attached to the posterior and right lateral walls of the nasopharynx and seemed to involve the right tonsillar fossa. It also extended upward to the sphenoid ridge on the right. The excised portions of tumor were classified as "nasopharyngeal fibroma" (fig. 2). Postoperatively he complained of intermittent obstruction to the right Eustachian

tube. Again, in retrospect, the removal probably was incomplete. By August 1952, the fullness in the right lateral pharyngeal wall area again was found and digital examination showed a firm mass measuring 1.5 by 2.0 cm. displacing the Eustachian tube and involving the upper tonsillar pole.

The symptoms of obstruction of the right Eustachian tube continued through January 1954. At this time irradiation, using a 2,000,000 volt therapy unit, was begun. A tumor dose of 6,000 roentgens was delivered in 31 days (16 treatments) "to a cylinder of tissue 7 cm. in diameter and 5 cm. in height," by Dr. Milton Friedman, New York City. The therapist estimated that the tumor had shrunk 60 per cent by volume and gave further treatment until a total tumor dose of 9,000 roentgens was delivered.

The patient returned on July 8, 1954 because of recurrent epistaxis. He reported some "regression" in the mass for two to three months, followed by recurrent growth. During this period of growth, he was being treated with large doses of androgens. A prominence now could be seen along the right lateral border of the soft palate displacing this structure downward. Because of continuing enlargement of the mass, it became necessary to extract three right upper molar teeth. Biopsy section again was reported as a benign nasopharyngeal angiofibroma (fig. 3). Following this procedure, the tumor began to fungate through this biopsy incision. On Nov. 11, 1954, the tumor was approached by splitting the lower lip and mandible in the midline, extending the incision upward and posteriorly in the gingivolingual gutter, elevating the mandible and cheek laterally to expose the pterygo-maxillary area. With this type of approach, the entire nasopharyngeal area could be visualized and explored adequately. A tumor mass measuring approximately 8 by 7 by 4 cm., filling the nasopharynx, displacing the soft palate, and reaching the base of the skull was removed. In this instance, all gross tumor was removed. This surgical specimen contained areas of fibrosarcoma in a nasopharyngeal angiofibroma. The details of the procedure in this and other cases will be presented at greater length in a paper in preparation by one of us.[5]

The patient's postoperative course was uneventful, except for the development of a small fistula posterior to the right upper gingival ridge extending into the nasal cavity, and some trismus. At present the patient is free of evidence of disease (May 1, 1955).

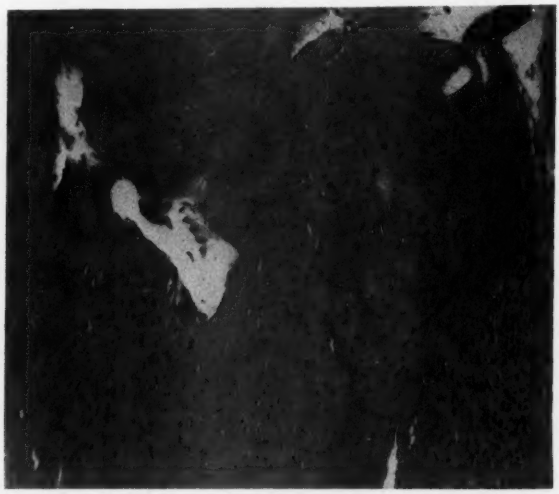

FIG. 3. Biopsy section following extensive external irradiation to the tumor; hyalinization increased over preceding figures, but note benign appearing stroma. ×100.

PATHOLOGY

The histopathology of the first three specimens in this case is similar to that described and illustrated in recent publications.[1, 8] In brief, as may be seen from the photomicrographs in this case (figs. 1 to 3, 6) the tumor is composed of two types of tissue; a vascular framework and fibromatous stroma. Sternberg[8] and more recently Allen[1] have emphasized the vascular component of this tumor in contradistinction to earlier reports which concentrated on the fibromatous elements. Both of these authors have commented on the close resemblance of this vascular element to arteriovenous aneurysms and Allen has recorded the almost equivalent picture in spider telangiectasis to the vascular areas in these tumors. Indeed, Sternberg regards the juvenile nasopharyngeal angiofibroma as a "distinctive variant of the angioma, with a unique stroma and stromal cell."[8]

Since the vascular element of the tumor was not the essential point of interest in the major specimen in this case, it will not be commented upon further.

The specimen removed at the last operation, consisted of an ovoid, bosselated mass of reddish-brown tissue measuring 8.3 by 7.1 by 4.1 cm. (fig. 4). The undersurface of the specimen had an area of mucosa (palate) measuring 3.6 by 2.3 cm. which showed a central zone of ulceration of 1.7 by 1.5 cm. in size and extended inward for a depth of 0.4 cm. The tumor mass was circumscribed and well de-

FIG. 4. Gross photograph of resected specimen 1954

marcated, although no true capsule was present. On section, the tumor was composed of resilient brownish-tan, reddish-pink tissue with small "cytsic" trabeculate spaces throughout and some small hemorrhagic foci near the middle of the mass (fig. 5).

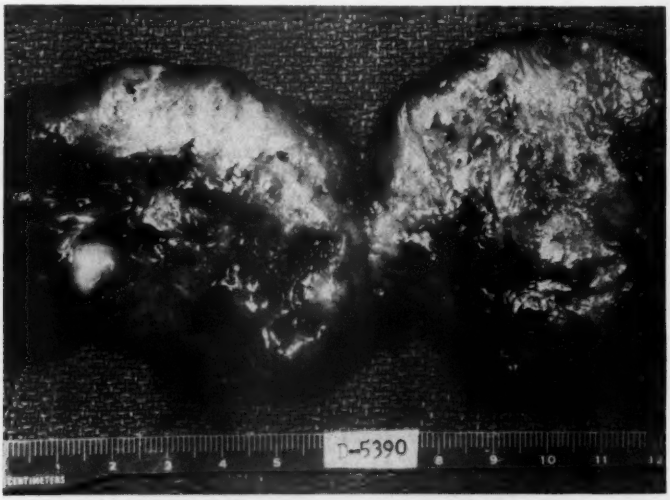

FIG. 5. Transected gross specimen showing vascular trabeculate stroma with foci of hemorrhage.

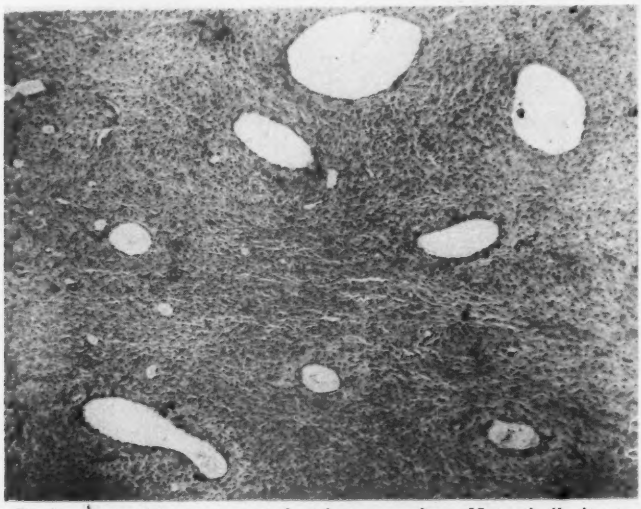

FIG. 6. Benign area of tumor resected at last procedure. Note similarity to preceding photomicrographs. This area merged gradually with those illustrated in the following figures. ×50.

Microscopically, the demarcation of the advancing edge of the tumor was as sharp as it was in the gross specimen because of the compression of the edge of the fibrous component coupled with engorgement of the vascular element at this site. Much of the tumor was in no way different from that seen in the earlier specimens and consisted of thin to thick walled vascular channels with a fibrous

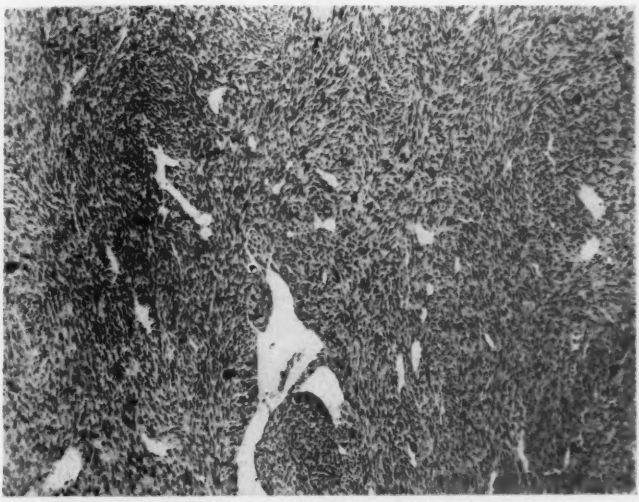

FIG. 7. Marked increase in cellularity, hyperchromatism and scattered tumor giant cells in this area of fibrosarcoma. Vascular framework still evident. ×100.

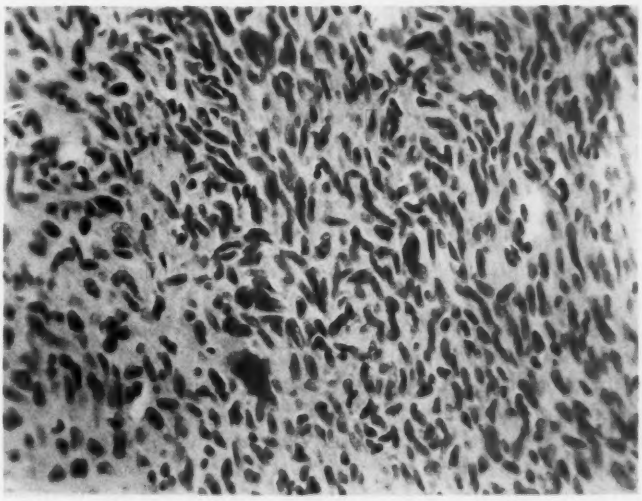

FIG. 8. Hypercellular area showing prominent nuclei, nucleoli, and tumor giant cells. ×225.

connective tissue matrix of fine and coarse collagen bundles interspersed with stellate connective tissue cells (fig. 6). As one approached the central portion of the tumor a marked increase in cellularity became apparent (fig. 7). Spindle shaped cells with prominent nuclei, nucleoli, increased mitotic activity, and finally numbers of bizarre tumor giant cells (figs. 7, 8) were evident. The vascular spaces were not obliterated, but on the contrary were accented because of the perithelial arrangement of tumor cells (fig. 7). Despite this, there was no breakthrough into vascular channels and none of the sarcomatous elements was found in vessels. Trichrome stains showed dense blue collagenous fibrils between the tumor cells. The diagnosis was areas of fibrosarcoma arising in a juvenile angiofibroma of the nasopharynx.

DISCUSSION

The fact that the tumor in this patient first became manifest at 48 years of age, suggests that the age-linked factor in "juvenile" nasopharyngeal angiofibroma may be more apparent than real. Others have made this suggestion previously. Its development in a male supports the contention of a sex-linked factor in this distinctive group of lesions. Even the best studied, purported case of nasopharyngeal angiofibroma in a female[3] is thought to be more suggestive of a nasopharyngeal polyp than of an angiofibroma.[1, 8]

The malignant change recorded in this tumor is of particular interest. While we shall be unable to answer those who say that this tumor was malignant from its inception and that the fibrosarcomatous portions were not biopsied in the three earlier attempts, it should be pointed out that no well documented cases of this change are recorded. This case cannot be documented as the first recorded instance of "spontaneous fibrosarcomatous degeneration" in this type of tumor, for to do so would be to neglect the triggering effect or actual induction of this change by the largest dose of external irradiation yet recorded for a tumor of this type. This change has not occurred too soon following irradiation therapy, as a bizarre radiation cancer which invaded the oropharynx and produced death in a 62 year old woman has been seen within 15 months after irradiation of the neck with 7,250 roentgens total tumor dose for follicular and solid carcinoma of the thyroid.[4] Many statements regarding the temporal relationships between exposure and the beginning of radiation cancer are not well documented or susceptible of proof, since one does not ordinarily take biopsy sections of these areas of prior irradiation until some new complaint or symptom is voiced or until some change becomes evident.

This case also suggests that the initial therapeutic approach to this type of tumor should be by some surgical method which affords the widest possible exposure of the entire nasopharyngeal area in preference to present technics via a transnasal or intraoral approach, both of which usually result in morcellation rather than excision. One such approach has been described by Kremens.[6] The operation employed in the present patient was similar, save that the mandible was divided in the midline to preserve the inferior alveolar nerve. Irradiation often is ineffective and can be followed by radiation osteomyelitis[8] or malignant change such as shown in the present report.

We do not know what the cellular changes portend in this instance, since no adequately documented cases of this type are available for comparison, nor can we anticipate what the late effects of this dose of irradiation will be. At present, the patient is free of evidence of disease, approximately seven months after surgery, and has returned to his usual way of life.

SUMMARY

A case of "juvenile" nasopharyngeal angiofibroma in a 54 year old man treated initially by partial surgical removal and extensive irradiation and subsequent complete surgical excision is presented.

Areas of fibrosarcoma were found in this previously benign tumor. The relation of these changes to "radiation cancer" is discussed briefly.

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APPENDICEAL FISTULA

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The term fistula is Latin in origin and means pipe. Dorland[4] defines a fistula as a deep sinus ulcer, often leading to an internal hollow organ. Appendiceal fistula is a communication between the cecum at the base of the appendix, and any other organ or structure, regardless of the etiology, providing the appendix has not been surgically removed. Appendiceal stump fistula is reserved for those conditions where a communication develops from the base of the appendix to an adjacent structure following surgical removal of the appendix.

Fitz[5] was well aware of the occurrence of appendiceal fistula. In 1886, he mentioned appendiceal fistula involving the deep circumflex iliac artery, which terminated fatally by exsanguination. Following Fitz's classical paper on appendicitis, medical authors[2, 3, 8, 10, 11, 13, 14] responded with many articles regarding typhlitis, perityphlitis, cecal granulomas and fibroplastic appendicitis.

Appendiceal fistula rarely is discussed in the current literature. Treatises on the diseased appendix and its complications have occupied less and less space in the medical literature as surgical interest has become concentrated along other lines.

Complications of the diseased appendix are seen with much less frequency today than formerly. This is due to the general acceptance of early surgery for the acute attack of appendicitis and heavy antibiotic therapy for any possible complication. However, particularly in the older age group, the signs and symptoms may not be classical and the attack not so acute. Add to this the use of antibiotics, and it is easy to see how an abscess might develop without a typical systemic response. Under these circumstances, the correct diagnosis of appendiceal abscess may be overlooked.

Appendiceal fistula can involve the gastrointestional tract—particularly the terminal ileum and ascending colon, the female genital tract, and the urinary tract. It is conceivable that appendiceal fistula may involve the biliary tract, as the authors have seen several acutely infected appendixes attached to the gallbladder by inflammatory adhesions. However, no such report was encountered in our review of the literature.

We would like to state at this point the purpose of this paper. It is to emphasize that any patient with a palpable lower abdominal mass, with or without an obstructive component, and/or a demonstrable fistula, must not be refused exploration because of the assumptive diagnosis of carcinoma with local inoperability.

Appendiceal fistula is uncommon. In 1951, Kelley[9] reported 4 cases of appendicoileal fistula and reviewed the literature to find only three references to

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the same condition. Garcia[6] has reported 1 case of congenital ileoappendiceal fistula.

PATHOGENESIS AND ETIOLOGY

Appendiceal fistula occurs following acute appendicitis with perforation and abscess formation. The attack of appendicitis may be acute and classical in all its manifestations, followed by perforation and abscess formation, or, it may be a mild inflammatory reaction about appendiceal fecaliths or foreign bodies—followed by a small leak with abscess formation. In any event, the local tissue response to the infection is the first step in fistula formation. The attempt by the body to localize and wall-off this leaking fecal stream calls for utilization of every structure and organ at its command, whether it be bladder, ascending colon, Fallopian tube, or small bowel. The attempt by the body to contain this infection also may be responsible for draining the abscess, thus the formation of a fistula. The iatrogenic element of heavy antibiotic therapy enters into the problem at times and fortifies the body in its attempt to localize the infection.

The etiology of appendiceal stump fistula is somewhat different than the above, in that a foreign body usually is the basis of the local abscess formation. In general, however, the inflammatory tissue response and the general pattern of fistula formation is the same. Wilson and associates[15] in describing 20 cases of nonspecific appendiceal granuloma, incriminated the residual stump of an incompletely removed appendix in 2 patients. Several authors have reported stump fistula developing around nonabsorbable suture material used by the surgeon at the time of appendectomy. Nemilov[12] thought that the use of nonabsorbable suture for both the stump ligature and purse string suture trapped mucosa between sutures and resulted in small abscess formation. Blegan and associates[1] reported 1 case of postappendiceal stump fistula present for 34 years.

DIAGNOSIS

As a rule, the correct diagnosis is not made prior to exploration. The usual preoperative diagnosis is carcinoma of the cecum. Many of these patients will have a palpable lower abdominal mass. The coexistence of cecal cancer and appendicular abscess must be kept in mind. Hellsten and Ramström[7] reported 7 cases, and were able to find 12 cases reported up to that time. Crohn's disease, mucocele, tuberculosis, and mycotic infection must be considered in the differential diagnosis. In most instances the fistula will not be demonstrated until the abdomen is open.

TREATMENT

The treatment is surgical intervention. The extent of surgery will be dictated by the structures involved, and the extent of the inflammatory response. In most instances, these patients will have had a bowel preparation prior to operation, and thus, a primary resection with an ileotransverse colostomy will be the operation of choice.

During the last year on the surgical service of The George Washington Uni-

versity Hospital, we have had 4 cases of appendiceal fistula. Each presented a different problem in diagnosis. The correct diagnosis was not made prior to exploration. The following preoperative diagnoses were made: 1) Terminal ileitis with ileovesical fistula; 2) Carcinoma of the cecum; 3) Endometrial carcinoma; 4) Carcinoma of the sigmoid with obstruction.

CASE REPORTS

Case 1. This 43 year old white woman was admitted to the hospital on Feb. 16, 1954, complaining of urinary frequency, dysuria, and pyuria of 11 months' duration. At the onset of her symptoms, she recalled a bout of diarrhea of short duration, otherwise, her past history was entirely negative. At cystoscopic examination prior to admission, a fistulous opening in the dome of the bladder was visualized. A barium enema (fig. 1) demonstrated a deformed cecum with an ileovesical fistula. Physical examination was negative except for minimal lower abdominal tenderness. The bowel was prepared and on February 18, an exploratory laparotomy was done. The preoperative diagnosis was terminal ileitis. At operation, a fistula was found between the base of the appendix and the dome of the bladder, with

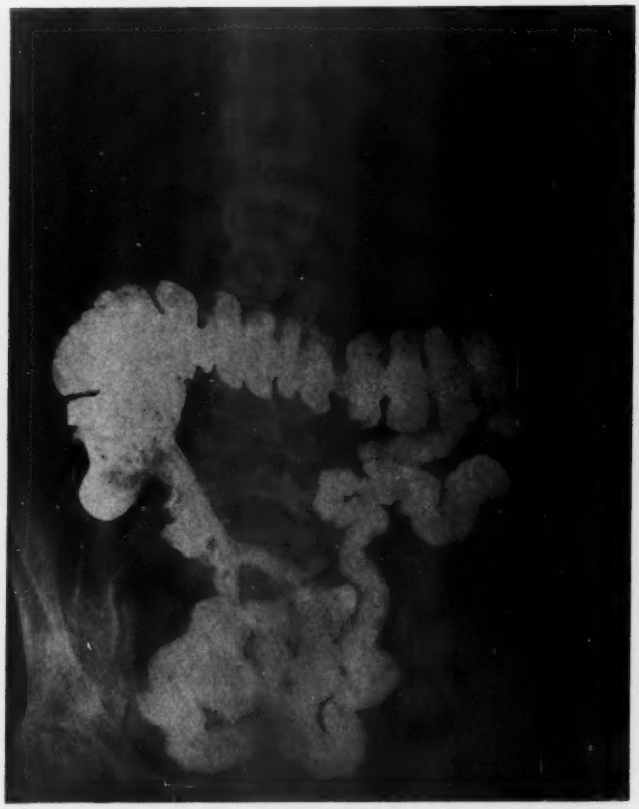

FIG. 1. Barium enema demonstrating a deformed cecum and an ileovesical fistula

marked inflammatory reaction surrounding the terminal ileum and cecum. The fistulous opening was excised and the bladder was closed. A right hemicolectomy and ileotransverse colostomy was done. She had an uneventful postoperative course and was discharged on March 2, 1954.

Comment: This patient was operated upon with the diagnosis of Crohns disease with ileovesical fistula. At the time of operation she was found to have an appendiceal fistula involving the bladder.

Case 2. This 70 year old white woman was admitted to the hospital on July 8, 1954, with the history of undue fatigue of one year's duration. Three weeks prior to admission she had a bout of cramping abdominal pain, nausea, and a chill of short duration. From that time until admission she was bothered by recurrent pain and nausea. A palpable abdominal mass was found upon physical examination. Barium enema (fig. 2) demonstrated a filling defect in the cecum and ascending colon. A diagnosis of carcinoma of the cecum was made. The bowel was prepared, and on July 10 exploratory laparotomy was done. At this time a *fist-sized* mass was encountered involving the cecum which had all the characteristics of malignancy. A right hemicolectomy was done with an end to end ileotransverse colostomy. When the specimen was opened, a retrocecal appendix was found which had perforated the

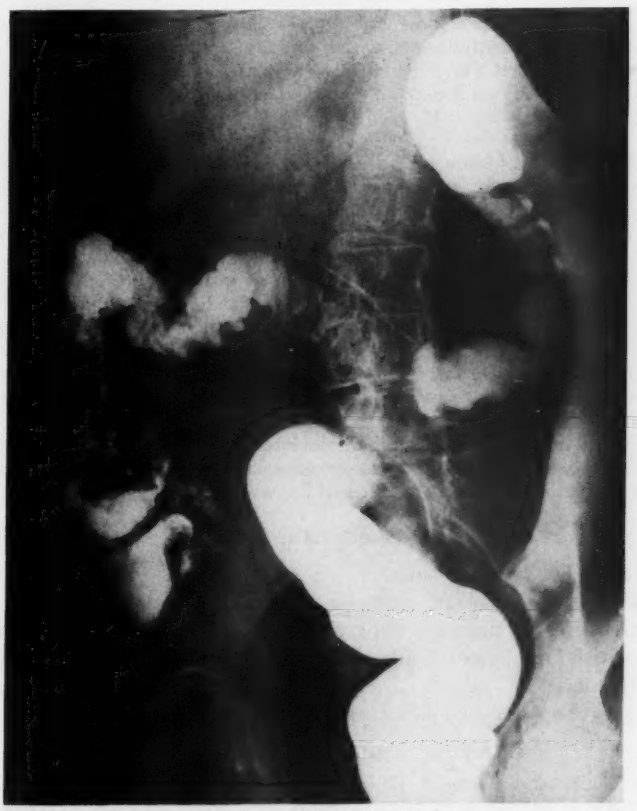

FIG. 2. Barium enema demonstrating a filling defect of the cecum and ascending colon

ascending colon 7 centimeters above the ileocecal valve. The tumor was composed of inflammatory tissue surrounding the fistula. The patient had an uneventful postoperative course and was discharged on July 20, 1954.

Comment: This patient was operated upon with a diagnosis of carcinoma of the cecum. The correct diagnosis was not made until the specimen was examined.

Case 3. This 68 year old white woman was admitted to the hospital for the first time on Nov. 26, 1954. A history on admission was that of a dragging right lower quadrant pain of one year's duration. Three days prior to admission the pain became more intense and was accompanied by diarrhea, nausea, and fever. Physical examination revealed bilateral lower abdominal tenderness without audible peristalsis. A roentgenogram of the abdomen showed a dilated small bowel compatible with paralytic ileus. A barium enema and intravenous pyelogram showed nothing abnormal. She was treated with antibiotics and discharged with the diagnosis of paralytic ileus—etiology unknown. On Dec. 21, 1954, she was readmitted because of recurrent lower abdominal pain, diarrhea, and a purulent vaginal discharge. A diagnosis of endometrial carcinoma was made. She refused further studies and was discharged. On Jan. 13, 1955, she was readmitted because of persistence of pain, chills, fever, and anorexia. Exploratory laparotomy on Jan. 20, 1955, showed a large mass in the right pelvis with an abscess cavity surrounding an appendiceal fistula. The fistula involved the right Fallopian tube. Appendectomy, right oophorectomy, and salpingectomy were done. The patient had an uneventful postoperative course and was discharged on Jan. 30, 1955.

Comment: This patient was explored with the preoperative diagnosis of endometrial carcinoma. At operation, she was found to have an appendiceal fistula involving the right Fallopian tube.

Case 4. This 58 year old white woman was admitted to the hospital on Dec. 15, 1954, with a three weeks' history of left upper quadrant abdominal pain. She had been treated with oral medications for *virus infection* for 72 hours. The week prior to admission she denied any bowel movements, but had passed flatus and some *whitish mucous*. Physical examination showed a distended and tense abdomen with right upper quadrant tenderness. A nontender pelvic mass was found by rectopelvic examination. It was thought that this patient had a carcinoma of the sigmoid with partial obstruction. The bowel was prepared and on Dec. 21, an exploratory laparotomy was done. At operation, an abscess cavity in the right lower quadrant was encountered which included the cecum and a portion of the terminal ileum. The appendix had been necrosed by the abscess. The perforated portion of the terminal ileum was resected and the abscess cavity was drained. She had an uneventful postoperative course and was discharged on Jan. 2, 1955.

Comment: A preoperative diagnosis of carcinoma of the sigmoid with partial large bowel obstruction was made in this instance. At operation, the patient was found to have an appendiceal fistula involving the terminal ileum.

SUMMARY

Appendiceal fistula is defined.

The pathogenesis, etiology, diagnosis, and treatment is discussed.

Emphasis is placed upon the danger of considering a lower abdominal mass a carcinoma with local inoperability, when a simple appendiceal fistula may be the cause of the mass.

Four cases of appendiceal fistula are reported.

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ANESTHESIA FOR THE POOR-RISK PATIENT

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It is becoming increasingly more difficult to define the poor-risk patient. With present day skilled internists, surgeons, and anesthesiologists it is rare to deny patients any necessary operations. It is, however, essential to evaluate the physical status of the patient prior to any surgical procedure. Reversible pathologic processes should be corrected and irreversible ones recognized. Thus, it is essential before any anesthetic is administered, to review the history, physical examination and laboratory data. There exists little doubt that surgery has been made safer in the poor-risk patient as the result of advancements in the field of anesthesiology.

The most important concern of the anesthesiologist is any reduction in functional reserve in the circulatory or respiratory systems. Reductions in circulatory reserve usually fall into two groups. First, those due to blood volume disturbances and, second, those due to cardiac disturbances. Blood volume disturbances may result from red cell deficiency, reductions in blood plasma or due to deficiencies of serum proteins. Any deficiency should be determined and treated preoperatively. It now is generally true that the blood volume replacement is carried out on the basis of hemoglobin, hematocrit and serum protein determinations plus clinical judgement. All of these may be greatly in error. Blood volume disturbances cannot be accurately diagnosed by the routine laboratory tests. The clinical application of blood volume determinations is well founded.[1] Very few hospitals are equipped to do these tests. Thus, management must be based on the history, laboratory tests and clinical judgement.

Red blood cell loss can be treated only by the use of red blood cells. In a 150 pound patient, 500 cc. of blood will increase the hematocrit 5 points, the red blood cell count 500,000 and the hemoglobin 8 per cent. In chronically ill patients who have suffered weight loss it has been shown by blood volume determinations that the hematocrit, red blood cell count and hemoglobin may be normal but with reduced total blood volumes. These patients may receive up to 40 cc. of whole blood per pound of weight loss prior to major surgery. The quantity of blood given depends upon the patient's physical condition, serum proteins, and cardiac status. We use the plasma expanders only in emergencies to maintain blood pressure in those patients for whom whole blood is not immediately available. The plasma expander may be used as a temporary measure while the blood is being typed and cross matched.

Patients with a reduction in blood volume are best managed with an inhalation anesthetic drug and with high concentrations of oxygen. Spinal anesthesia is contraindicated due to the paralysis of the vasoconstrictor fibers producing a

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vasodilatation and thus a greater discrepancy between vascular capacity and blood volume which further accentuates the shock. In our hands the use of ether-oxygen anesthesia has been found to be safe. With the use of ether, high concentrations of oxygen may be used. Other anesthesiologists prefer cyclopropane-oxygen combinations in shock patients. Pentothal® should be used cautiously in patients with reduced blood volume because of its adverse circulatory effects.

There is no condition that requires closer cooperation between the internist, surgeon and anesthesiologist than the surgical patient with heart disease. Generally speaking, it may be said that the patient with cardiac disease who carries on his daily routine without disability can be given an anesthetic without any increase in risk. Patients who complain of precordial distress, dyspnea on exertion, palpitations, and dependent edema, thus indicating a possible decrease in cardiac reserve, should have the benefit of a preoperative evaluation of cardiac status. Surgical patients with hypertension, coronary artery disease, myocardial failure, arrhythmia, or congenital cardiac lesions should be carefully managed.[2]

The patient with hypertension without associated cardiovascular complications should have no increase in surgical or anesthesia risk. However, pathologic complications may be difficult to evaluate. Such complications as arteriosclerotic changes in the brain or kidneys, associated coronary artery disease and myocardial damage all may increase the mortality rate during any surgical procedure. It is for these reasons that such patients should be evaluated preoperatively and spinal anesthesia should be avoided. This is due to the hypotensive effect of spinal anesthesia which may not be controlled as rapidly as desired by use of the vasopressors. Another problem that we have seen in this group of patients is the use of the antihypertensive drugs in the control of elevated blood pressures. We have had 20 surgical patients receiving one of the Rauwolfia group of drugs for the treatment of hypertension. Five of these had a marked fall of blood pressure with an associated bradycardia. Two of these patients were treated with Antrenyl®, a vagal-blocking agent, with a beneficial response.

In reviewing these cases it appears that this is a vagal stimulation. This response appears to be enhanced when the parasympathetic portion of the autonomic nervous system predominates such as under spinal anesthesia or when using a parasympathomimetic drug such as Pentothal®. This also may apply to other drugs such as cyclopropane, but we have had no experience with this drug in this group of patients. It is recommended that patients on antihypertensive drug therapy should discontinue use of the drug at least two weeks prior to an elective surgical procedure. In an emergency surgical procedure, a vasopressor drug should be immediately available to control any depression in blood pressure that may occur and a vagal-blocking drug should be given.

An increasing number of patients with coronary artery disease are having surgical operations. This probably will be an ever increasing problem due to an increase in the life span, and an increased life expectancy in patients with proved coronary disease. In 1930, Butler, Feeny, and Levine[3] reported a mortality rate of 7.7 per cent in operations upon patients with angina pectoris and a mortality

of 44.5 per cent in patients with previous coronary thrombosis. Levine[5] recently emphasized that this study no longer reflects the true risk of this group of patients. Recently studies by Morrison,[7] Hannigan,[4] Lockhead,[6] have reported an average mortality rate of 6.3 per cent in the anginal group and 7.2 per cent in the coronary thrombosis group. Following an acute myocardial infraction, it generally is agreed that from three to six months should elapse before any elective surgical procedure is planned in order to permit the myocardium to heal.

Hypotension and hypoxia should be avoided in the management of anesthesia in this group of patients. Falls in blood pressure should be prevented. Thus, spinal anesthesia is not the choice for the patient with coronary artery disease. In the event of a blood pressure fall appropriate therapeutic measures should be immediately started. These consist of blood to replace any blood loss during operation, vasopressors for the treatment of neurogenic shock, and vagal-blocking drugs to combat vagal-traction reflexes occurring during surgery. In chronically ill patients, adrenal cortical insufficiencies may exist. The use of cortisone preparations have been helpful. To avoid hypoxia high oxygen concentrations are used in the anesthetic mixtures. Respiratory obstructions, should they occur, must be immediately corrected. Positions on the operating table that reduce respiratory exchange should be avoided, and if they occur respiratory assistance should be given. The use of any anesthetic drug which produces respiratory depression, especially the muscle relaxants, may necessitate respiratory assistance. Excitement and exertion should be eliminated to prevent an increase in oxygen demands. Tachycardias and arrhythmias should be avoided because of the danger of increasing the oxygen demand of the myocardium. To minimize the excitement and thus eliminate this cause of hypoxia, a small quantity of Pentothal® is given in the patient's room. We prefer Pentothal® inductions plus ethylene-oxygen and then ether-oxygen maintenance for patients with coronary artery disease.

We are more concerned with the surgical patient in myocardial failure than any other cardiac patient. Any patient who presents the symptoms of dyspnea, swelling of the ankles, or a history of previous heart failure should be managed as a heart failure case. Only in the rarest of emergencies should a patient in heart failure be given an anesthetic. With the rapid methods of intravenous digitalization, heart failure can be successfully treated within a few hours and a surgical procedure then can be more safely carried out. In this group of patients the anesthesia of choice generally is regional anesthesia. In abdominal procedures we prefer spinal anesthesia because it acts as a bloodless phlebotomy by producing a peripheral vasodilatation allowing the blood to pool in the periphery, thus reducing the venous return to the heart and pulmonary bed.[9] This reduces the load on the heart. These patients should be given small doses of spinal anesthetic drugs to avoid high levels of anesthesia and intercostal paralysis which may interfere with respiratory exchange. In addition, continuous administration of oxygen should be used during the procedure. Utmost care should be taken to avoid overloading the circulation. This is done by limiting the quantity of intravenous fluids and by replacing only that quantity of blood that is lost during

surgery. The various positions on the operating table that interfere with respiratory exchange or increase the venous return to the heart should be avoided. Such positions as the steep head down are not tolerated by the patient with myocardial damage. The prone position without adequate respiratory support reduces the respiratory exchange preventing adequate oxygenation of the patient. Myocardial failure occurring in the thyrocardiac patient should be treated by the antithyroid drugs because digitalis preparations are of little value in the treatment of this particular type of cardiac failure.

There are two groups of arrhythmias which concern us in anesthesia. These are heart block and multiple premature ventricular contractions. Any delay in cardiac conduction during anesthesia may, in the presence of shock or hypoxia, result in complete heart block. Patients with heart block cannot be benefited by preoperative preparations. They do, however, require careful management during the operative procedure. Multiple premature ventricular contractions may develop into ventricular fibrillation during operation. These patients can be prepared preoperatively by the use of either procaine amide or quinidine. It is preferable to use this preparation prior to operation because of the possible toxic effects of these drugs that can be better controlled in the conscious patient. Cardiac asystole or ventricular fibrillation is cardiac arrest so far as cardiac output is concerned and necessitates prompt cardiac resuscitation.

There are three valvular defects that concern the anesthetist. The surgical patients with mitral stenosis, aortic regurgitation, and congenital heart lesions should be carefully evaluated and managed during the course of anesthesia. In mitral stenosis, tachycardia must be prevented because of the occurrence of pulmonary edema. The belladonna group of drugs are omitted in the preoperative medication.[8] If, during the course of anesthesia, tachycardia developes, physostigmine may be beneficial. Aortic regurgitation patients, due to associated coronary insufficiency, should be managed the same as patients with coronary artery disease to prevent hypoxia and a fall of blood pressure. Patients with congenital heart lesions usually are chronically hypoxic. Many of these patients are children, and crying or struggling during the induction of anesthesia will further increase the degree of hypoxia. Many of such patients are apprehensive. The apprehension and fear associated with an anesthetic procedure will increase the intrinsic epinephrine blood levels. This combined with hypoxia sets the stage for ventricular fibrillation. In this group of patients it is best to avoid any of the cardio-toxic anesthetic drugs. The use of intravenous Pentothal® or rectal Avertin® as a basal anesthesia in the patient's room has done much to make surgery safer for these patients. All patients with reduction in circulatory reserve are connected to the operating room cardioscope during the course of anesthesia and surgery so that disturbances in cardiac conduction can be recognized immediately.

RESPIRATORY REDUCTIONS

Reductions in respiratory reserve are due to respiratory obstructions and a decrease in respiratory surface. Obstructions in the airway may be due to aspira-

tion of regurgitated gastric contents, purulent secretions, blood in the tracheobronchial tree, bronchospasm, tumors of the air passages, and mechanical obstruction of the upper airway. Aspiration may occur in anesthetizing patients with food in their stomachs, intestinal obstruction, or bleeding peptic ulcers. This complication can be prevented by intubating these patients under topical anesthesia. The aspiration of regurgitated gastric contents can be prevented by using a cuffed endotracheal tube. Purulent secretions in the air passages may be the result of chronic bronchitis or bronchiectasis. Blood in the airway may be the result of adenomas, tuberculosis or carcinoma of the respiratory tree. The wet cases of bronchiectasis are now rare due to the use of antibiotics in the preoperative preparation of these patients. It may be necessary in the rare patient that comes to surgery with a copious quantity of secretions to use either endobronchial anesthesia or to block the bronchus from which the secretions are being liberated. This is done to prevent contamination and obstruction of the uninvolved side. Bronchospasm as seen in the asthmatic patient now is being treated preoperatively with either cortisone or ACTH. These patients are best managed by a basal dose of Avertin®, avoiding Pentothal®, and using ether-oxygen for maintenance. Pentothal® is a parasympathomimetic agent and its use is avoided in the severe asthmatic. Tumors obstructing the air passage may be either intrinsic or extrinsic. Intrinsic tumors may be scar tissue masses, tuberculous granulomas or adenomas. The extrinsic tumors most often seen are substernal thyroid adenomas or aneurysms of the arch of the aorta. Some of these obstructions can be relieved by an endotracheal tube. In others, an endobronchial anesthetic can be given through a tracheostomy opening. Some of these patients are benefited by lightening the density of the anesthetic mixture by using helium. Mechanical obstructions of the upper airway may be due to nasal secretions, tongue obstructions, relaxation of the lower jaw, excessive salivation and laryngospasm. These usually are technical in nature and can be controlled by the anesthesiologist.

Reductions in respiratory surface can be measured preoperatively by the various pulmonary function tests. A fair estimation can be obtained by questioning the patient as to the amount of exertion necessary to produce dyspnea. Reductions in pulmonary reserve may result from collapsed lung due to pneumothorax, hydrothorax, empyema, thoracoplasty, or atelectasis. Loss of respiratory surface may result from previous pulmonary resections, emphysema, pulmonary edema and tuberculosis.

During the course of surgery, some of the surgical positions such as the steep head down, the prone position, and the lithotomy position may reduce the vital capacity. In this group of patients, regional anesthesia is the choice whenever it is thought that it will produce an adequate degree of anesthesia for the surgical procedure. If general anesthesia becomes necessary, mixtures containing high oxygen concentrations should be used. Any reduction in tidal exchange should be assisted either by manual compression of the rebreathing bag or by machines that will automatically support respiration. The steep Trendelenberg position should be used only when absolutely necessary and for as short a period of time

as possible. In the prone position, the patient should be supported on pillow rolls in order to allow for anteroposterior expansion of the thorax.

The poor-risk patient generally is given an anesthetic that is least disturbing to his physiologic processes. This does not mean that less potent anesthetic drugs should be used, thus reducing oxygen concentrations and producing inadequate anesthesia. This results in anesthesia which now has to be supplemented under adverse circumstances in order to complete the surgical procedure. Thus, insufficient anesthesia is not the answer in the management of the reduced resistence of the poor-risk surgical patient. Select the safest anesthetic that will meet all the requirements of the surgical procedure.

SUMMARY

Some of the poor-risk patients have been discussed and their management outlined. In the selection of anesthesia for these poor-risk patients the reduction in their physiologic reserve should be considered. The final choice of anesthesia should be the decision of the anesthesiologist. This decision is made after consulting with the internist concerning the physical status of the patient and the surgeon regarding the requirements for the surgical procedure.

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MASSIVE HYPERTROPHIC GASTRITIS: REPORT OF THREE CASES

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Massive hypertrophic gastritis is a rare pathologic condition of the gastric mucosa. It may be localized or generalized. It may be unassociated with other gastric disease or it may be seen with existing gastric ulcer, polyps or malignancy. The relationship, if any, to polyposis is unknown as is the relationship to malignancy.

Balfour[1] encountered only 1 case in 8,000 gastric operations. Eliason and Wright found only 1 in a similar number of autopsies. Schindler and McGlone[12] reported 3 cases, observed gastroscopically. Maimon, Bartlett, Humphreys and Palmer[7] reported 6 in 5,765 gastroscopic examinations and 4 diagnosed by roentgenographic and gastrocopic findings.

The etiology is unknown.[1,15] Localized massive hypertrophic gastritis is seen in association with gastric ulcers and in localized areas in gastric malignancy. It may be present in a generalized form, and usually is associated with increased gastric acidity.

There are no specific symptoms. The complaints frequently are those of hyperacidity, epigastric distress relieved by alkalies, fullness, bloating, and epigastric discomfort sometimes ulcer-like in character. Eructations, nausea, vomiting, and weakness may be present.

The roentgenographic appearance (fig. 1) is that of giant rugae frequently most prominent along the greater curvature. These folds frequently resemble polyps or malignancy from which they always cannot be distinguished.

The pathologic changes in the stomach in this condition are striking. The inner surface of the stomach has been likened in appearance to the brain with pronounced convulutions, and the stomach feels like *a bag of worms* or *resembling a huge varicocele*. Grossly, (fig. 2) the mucosa is composed of thickened rugae which persist even when the stomach is distended or the wall stretched. This striking picture appears as a massively exaggerated form of the entity commonly described as chronic hypertrophic gastritis. When the thickened soft gastric wall is opened, it is found to be freely movable over the underlying muscularis and serosa. Microscopically, (fig. 3) the deepened convulutions are found to result from hypertrophied gastric glands with infiltration of the stroma by lymphocytes, plasma cells and eosinophils. There is an increased number of hyperplastic lymphoid follicles. Frequently, the gastric glands are dedifferentiated and resemble intestinal mucosa. Grossly, there may be polypoid formations on the rugal folds, indistinguishable grossly from true polypoid gastritis. Microscopically, however, the submucosa does not develop into a fibrous core as

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FIG. 1. Roentgenologic examination of the stomach reveals the typical appearance of giant rugae.

in the polyp and the muscularis does not enter the stalk as seen in true adenomatous polyps. Malignant change in true adenomatous polyps is common, yet the relationship between the polyp-like formation in massive hypertrophic gastritis and malignancy is not clearly established. True evidence is lacking that the polypoid formation in massive gastritis develops into true adenomatous polyps.

The diagnosis depends on the roentgenographic appearance of the gastric mucosa, and on gastroscopy. However, frequently this condition cannot be distinguished from malignancy by either of these methods. The diagnosis may be established only after laporatomy and gastric biopsy.

When the diagnosis is confirmed, treatment will depend upon the severity of the symptoms, the extent of the lesion, and its association with other pathologic conditions within the stomach. It will vary from biopsy to subtotal resection.

CASE REPORTS

Case 1. A 69 year old white woman was hospitalized because of urinary distress of three years' duration. Moreover, for seven months she had noticed increasing anorexia, nausea

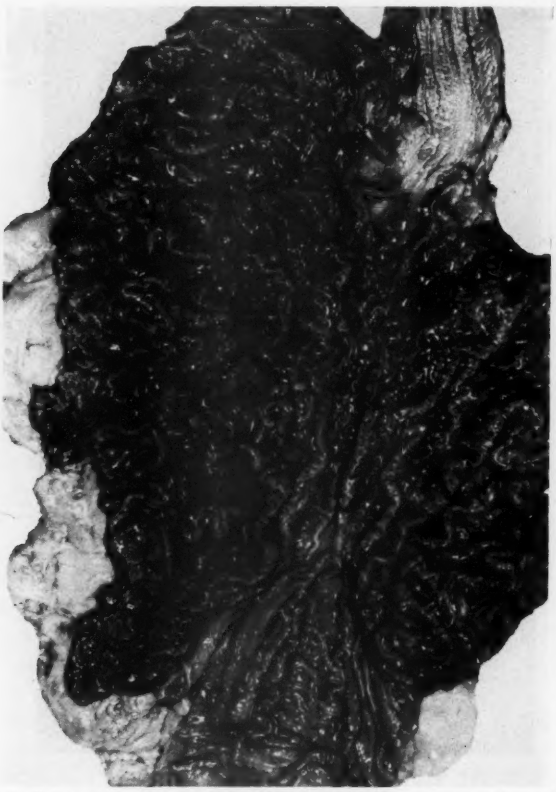

FIG. 2. Gross appearance of stomach showing giant rugae throughout

and upper abdominal fullness. She had lost 13 lbs. in weight during a 12 month period. In spite of these symptoms, she had no vomiting, melena, jaundice, or acute pain.

Physical examination showed a pale, elderly woman with evidence of recent weight loss. Examination of the heart, lungs and extremities disclosed no abnormalities. The abdomen was soft and nontender. The liver edge was palpable 3 cm. below the costal margin. No masses were detected.

Roentgenologic studies. Roentgenograms of the chest were normal except for fibrotic changes in both lungs. An intravenous pyelogram showed dilatation of the left ureter, cause undetermined. An upper gastrointestinal roentgenologic study showed marked enlargement and distortion of the mucosal folds of the stomach with apparent prolapse of the gastric mucosa into the duodenal bulb. There was no six hour gastric retention. The impression from the roentgenograms was that of distortion and hypertrophy of the gastric rugal folds most likely due to neoplastic infiltration of the stomach. The possibility of unusually severe gastritis accounting for the findings could not be ruled out.

Upon gastroscopic examination the angulus and prepyloric area were visualized. The mucosa appeared grossly hypertrophied, swollen, congested and edematous. There were areas of polypoid hyperplasia with adenomatous overgrowth. The remainder of the stomach mucosa had the same appearance. Impression, "polypoid hypertrophic gastritis possibly with malignant changes." A laparotomy was advised.

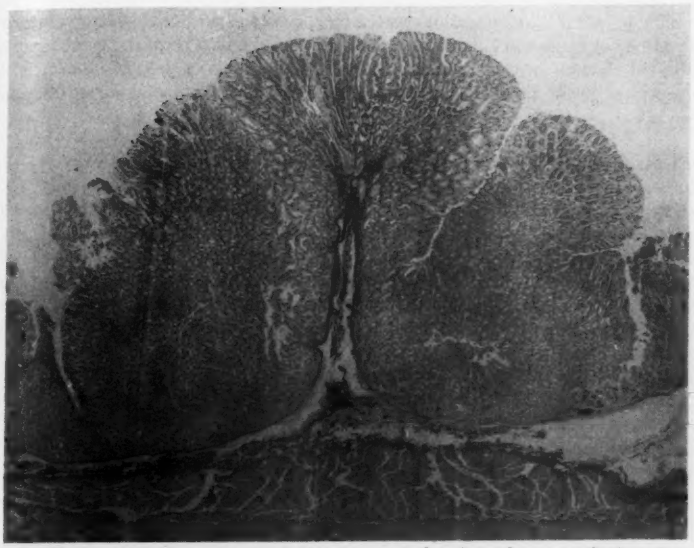

FIG. 3. Microscopic appearance of giant rugae showing the convolution formed by hypertrophic gastric glands.

At operation, the stomach was found to be thick-walled and the serosal vessels were dilated. It seemed to be full of soft material as though it contained coils of angleworms. On opening it, the mucosa was greatly hyperemic and there were innumerable polypoid projections which were friable and bled easily. This condition prevailed throughout all of the gastric mucosa. It was less noticeable, however, near the cardiac end. The lower ¾ of the stomach, therefore, were resected including the first part of the duodenum. The postoperative course was uneventful.

Pathologic Examination. (a) Gross description. The specimen consisted of the body of the stomach and pylorus with attached greater omentum. The lesser curvature measured 17 cm. and the greater 32 cm. in length. The serosa was pinkish-grey, smooth and glistening. The wall was thicker than normal. The mucosa was generally polypoid and at the duodenopyloric junction measured 2 cm. in thickness. The duodenal mucosa was glistening and coarsely granular. (b) Microscopic examination showed evidence of gastric polyposis with massive hypertrophic gastritis.

Case 2. This case is presented with permission of Dr. Calvin Klopp.

The patient, a 32 year old white man, was hospitalized with complaints of flatulence, epigastric burning discomfort and occasional vomiting, of three months' duration. His distress did not seem to be related to food but occasionally awakened him at night. He vomited from time to time and this, or the use of antiacids, relieved his discomfort. There were occasional remissions lasting from two to three weeks. There was no history of melena or hematemesis and no weight loss. He had been known to have diabetes for eight years.

The only positive finding on physical examination was moderate epigastric tenderness on deep palpation. Distention, masses or other abnormalities were not detected.

Upper gastrointestinal roentgenograms showed a filling defect, probably neoplastic, in the upper third of the stomach.

At operation, all organs were grossly normal except the stomach. There was evidence of diffuse thickening, especially in the region of the cardia. The stomach was opened and re-

vealed multiple, villus like protrusions on the mucosa of the greater curvature of the cardia. A biopsy of one of these was taken and frozen sections made. The pathologist reported benign polypoid changes in the stomach and made a diagnosis of polypoid gastritis. In the pyloric region of the stomach there were many greatly hypertrophied rugae. The stomach was closed. The postoperative course was uneventful.

Case 3. Massive hypertrophic gastritis in this case was discovered at autopsy. The patient was a 60 year old white man that had been admitted to the hospital many times. He had a vague story of anorexia and on his previous admission had complained of severe intermittent indigestion like *gas pains* radiating through to the back, but not elsewhere, of 24 hours duration. On admission, he was thought to have an acute gastritis. On his first admissions he was known to have Laennec's cirrhosis, and his gastric complaints always had been attributed to this condition. He had not had a gastric roentgenogram or gastric analysis. He died as the result of a pulmonary infarction, congestive heart failure, and hypertensive arteriosclerotic heart disease.

At autopsy, the stomach mucosa throughout was thrown into exaggerated rugal folds, and the mucosal epithelium was hyperplastic. There was a moderate dense chronic inflammatory infiltrate consisting chiefly of lymphocytes, plasma cells, and eosinophils scattered throughout the mucosa and submucosa and to a lesser degree in the muscularis. Several lymphoid accumulations also were present in the mucosa and submucosa.

SUMMARY

A rare condition of the gastric mucosa characterized by giant rugal formation has been reviewed with the addition of 3 cases. The physical appearance which it sometimes assumes, may be indistinguishable grossly from polypoid gastritis or malignancy. However, the conditions although indistinguishable roentgenologically or gastroscopically, can be differentiated by microscopic examination. Treatment varies from biopsy alone to conservative resection.

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SUBSTITUTE POUCH FOLLOWING TOTAL GASTRECTOMY

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Increasing knowledge of gastrointestinal physiology suggests that the postgastrectomy syndrome may be theoretically alleviated.[3, 4, 9] Recognizing the unpleasant effects of the dumping syndrome, [1, 7] some surgeons have resurrected the Billroth I procedure. It was hoped that by restoring the natural gradient of descent in the alimentary tract, rather than by gastrojejunostomy, the unpleasant effects of the dumping syndrome could be avoided.

Removal of the entire stomach is considered necessary in certain conditions such as linitis plastica, multiple ulcers, polyps, carcinoids of the stomach, and diffuse hemorrhagic gastritis. Other recorded indications for total gastrectomy are not shared by all surgeons.

Restoration of continuity of the gastrointestinal tract following total gastrectomy generally requires some form of esophagojejunostomy. Although esophagoduodenostomy is feasible, it becomes exceedingly difficult when portions of the lower esophagus must be resected. In such cases the Roux en Y procedure or an isolated segment of the jejunum has been used. Reports by Longmire,[6] Hunnicutt,[2] Lee,[5] State,[8] and Watkins[10] and others have shown that it is possible to interpose a segment of transverse colon as a reservoir following total gastrectomy. This has several appealing aspects: It may be possible by this method to restore the continuity of the alimentary tract in the proper fashion. It permits the temporary storage of food in the reservoir. To some degree, the peristaltic motions of the new stomach may simulate the churning action of the stomach. Furthermore, by mixing with the secretions in the duodenum, some of the initial digestive processes such as emulsification of fats, is begun. When this *mixed* food enters the jejunum no irritable reflex is initiated as with other types of repair. Therefore, the postgastrectomy syndrome (variously thought to be due to rapid distention of the jejunum or to heterotonic concentrations of the fluids ingested) conceivably may be alleviated or even forestalled. This syndrome has for its characteristics some combinations of fullness, hyperperistalsis, vertigo, fainting, clammy sweats and diarrhea. The stool often contains unabsorbed fat and nitrogen. This condition, when persistent or left uncorrected, may lead to anemia, inanition and eventual death. Obviously, this sometimes can be worse than the original disease itself. It has been shown in dogs[3] that nitrogen in the stool is considerably less after total gastrectomy if the anastomosis permits food to pass through the duodenum. Therefore, any method of alleviating such potential symptoms is appealing and worthy of consideration.

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The following is a description of technic and a resume of 3 cases in which the transverse colon was used to replace the stomach following total gastrectomy.

OPERATIVE TECHNIC

An operative procedure of this scope requires adequate exposure. Since it often is necessary to remove a segment of lower esophagus, yet at the same time be able to visualize the duodenum, it is believed that a long left thoracoabdominal incision is best. The diaphragm is split radially and the lower esophagus is mobilized. Gastrectomy is done with resection of additional tissues and organs as necessary. Following this procedure the transverse colon is elevated and mobilized. At the splenic and hepatic flexures, the transverse colon is divided. The intermediate segment of colon is anastomosed, in an antiperistaltic manner, to the resected ends of duodenum and esophagus respectively. It is imperative that the mesentery of this segment bearing the important middle colic artery be handled gently, for the success of the transplant depends upon the integrity of this blood supply. The upper esophagocolic anastomosis may be above or below the diaphragm depending upon the extent of esophageal resection (see inset in illustration). Following the completion of these anastomoses, the ends of the colon are brought together and anastomosed and the mesentery repaired. All potential openings in the mesentery are obliterated.

A Levine tube is placed down through the esophagus into the transplant for drainage purposes. Suitable drains are placed in proximity to the anastomoses.

The postoperative care is no different than with any other major abdominal case with one exception. The patency of the Levine tube *must* be maintained; this depends on aggressive action by the nurses to remove the thick mucoid secretions from the transplant. These secretions can easily plug the tube during the critical postoperative period when distention and regurgitation could be disastrous.

CASE REPORTS

Case 1—H. L., this 64 year old white man was admitted to the hospital on April 15, 1954. Seven weeks before admission he began to lose his appetite and to tire easily. Shortly thereafter he had a tarry stool every day for four days and lost approximately 10 pounds. He also regurgitated a small quantity of food. He has had a mild case of diabetes for the past 15 years and recently has been taking 15 to 20 units of Insulin daily. Four years before admission a roentgenologic gastrointestinal study done at another hospital was thought to show an old gastric ulcer. He had an esophagoscopsy at that time and nothing abnormal was seen. He had had two previous operations for hemorrhoids. The past history was otherwise essentially negative.

Physical examination on admission showed a rather pale, somewhat obese, white man, in no acute distress. The head and neck were essentially normal. His heart and lungs were clear to auscultation and percussion. His blood pressure was 122/68. The abdomen was soft and nontender with no palpable masses. The remainder of the examination showed only a small prolapsed hemorrhoid.

Laboratory studies on admission shows serology negative. The hematocrit was 42, white blood cells 8,050 per cu. mm. and total protein 6.7 with a normal A/G ratio. The fasting blood sugar was 170 mg. per cent and serum chlorides, alkaline phosphatase, blood urea nitrogen and CO2 were essentially normal. Gastric analysis shows free HCl rising as high as 104 units after histamine.

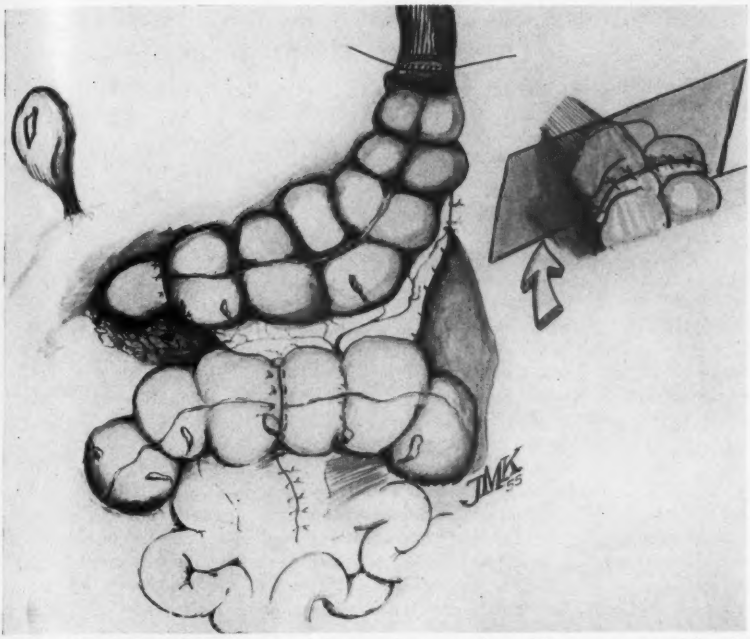

FIG. 1. Sketch depicting aggregate of operative procedures. Inset at upper right shows colon traversing plane of diaphragm with proximal anastomosis (case 1) at level of inferior pulmonary vein.

Roentgenogram of the chest showed increased markings in both upper lung fields which were apparently due to an old healed tuberculosis. Barium enema showed the colon essentially normal. Barium swallow showed partial obstruction of the esophagus at its junction with the cardia and some irregularity of the distal end of the esophagus. Repeat esophagoscopy on two occasions was negative for pathology. Accordingly on April 28, through a thoracoabdominal approach, a large carcinoma of the cardia was palpated. This extended inferiorly and posteriorly to the tail of the pancreas. There were no palpable enlarged nodes along the lesser curvature. There was no palpable metastasis to the liver. In view of the good curative prospects, a total gastrectomy, including en bloc resection of the lower one-third of the esophagus, spleen, distal pancreas and greater omentum was done. The remaining end of the esophagus appeared to be free of malignancy. However, an additional small rim of esophagus was given to the pathologist for frozen section. No malignancy was seen. Accordingly a segment of transverse colon was interposed in an antiperistaltic fashion.

The postoperative course was satisfactory. The patient graduated from liquids to a soft diet. To this were added vitamins and ferrous iron. On a subsequent examination gastrointestinal studies showed a questionable small abscess with a fistulous tract beside the upper anastomosis. This, however, had disappeared on a later examination on June 30. At the present time (May 15, 1955) the patient has gained five pounds and is eating well. He complains of an occasional tendency to regurgitate, especially when reclining. His diabetic status is controlled.

Comment: This is a gratifying result in an elderly patient. Fluoroscopy on several occasions showed that the antiperistaltic transplant showed isoperistaltic waves. The significance of this is not clear. The weight gain was unexpected. This has been an encouraging finding.

In this patient the *dumping syndrome* has been successfully averted. It is believed that

the reservoir prevents the sudden descent of food into the jejunum, which prevents both irritation and distention.

Case 2—I. C., this 54 year old white man was admitted with a chief complaint of fainting of two hours' duration. He had diabetes which was regulated by diet. Nine years previously a pancreatic cyst had been removed, and three months later a fistulous tract was excised. Seven years ago he had one episode of massive hematemesis which subsided with conservative therapy. A gastrointestinal roentgenologic study then was without significant findings. A gastroscopy showed a small puckered area in the stomach. He was relatively well until two hours prior to admission when he was awakened at 1:00 a.m. with a feeling of faintness and profuse sweats. While reaching for a glass of water, he fainted and was unconscious briefly. On the second attempt to sit up, he fainted again. On the night prior to admission he had had a normal bowel movement. No bloody or tarry stools had been noted. He complained of slight nausea, but no vomiting. There was a mild ache in the right lumbosacral area on the day prior to admission.

The patient was a well developed, well nourished white man, who was pale, perspiring and apprehensive. The blood pressure was 110/70, and pulse 72. The skin was cool and moist. The pupils reacted normally. The heart and lungs were normal. Abdominal examination and rectal examinations did not disclose anything of importance.

Laboratory studies on admission were as follows: Hemoglobin 12.4 Gm., hematocrit 44, and white blood cells 16,900 per cu. mm. The urine sugar was 4 plus. No blood dyscrasias could be found. The patient was transfused on admission. The stools were 4 plus for occult blood. At 5:30 a.m. on the morning of admission the patient vomited 500 cc. of dark red blood with a rapid drop in blood pressure to 70/40. Transfusions were continued. On the following day, he continued to vomit small quantities of blood, but the pressure and pulse remained stable until 12:30 p.m. on April 27. At this time he vomited 750 cc. of dark blood and promptly went into shock. He was taken to the operating room where a laparotomy was done. The duodenum was opened and found free of lesions. The stomach was opened, and handfuls of clots were removed, but no lesion could be found. No varices were noted. The lower two-thirds of the stomach was excised and a Billroth 1 anastomosis was made. There was no lesion in the specimen. The patient was returned to the ward in good condition. The diabetic status was controlled by Insulin, as indicated. He was placed on antibiotics and Vitamin K. The first 24 hours were not remarkable. On April 28 he began to bleed and blood passed back through the Levine tube. He immediately was given 1,000 cc. of whole blood. The hematocrit was 35. On April 29 a Blakemore tube was inserted, but the bleeding persisted. This pattern of bleeding (and replacement) continued unabated until May 5. During this interval there was an elevation of temperature to 40 C. with disorientation which responded to Aspirin and iced alcohol baths. On May 5 the patient was explored and through a left thoracoabdominal incision the remaining portion of the stomach was resected. Pathologic examination showed an acute erosive hemorrhagic gastritis. The spleen was excised. A segment of transverse colon was brought up and interposed between the esophagus and duodenum. On May 7 the condition of the patient was satisfactory except for occasional complaints of shortness of breath. Later, in the same day he began to cough frequently and expectorated brown viscous material. Following the episode of coughing, his condition rapidly deteriorated and he died at 11:00 a.m. on May 8, 1954.

Comment: In retrospect, it is believed that this patient died from massive aspiration of regurgitated material from the esophagus and transplanted colon. Emphasis is placed on maintaining the patency of the decompressive tube. As previously stated, mucus plugs from the pouch frequently occlude the lumen. This precedes reflux of the bloody mucoid material past the obstructed Levine tube. This reflux is enhanced unless the patient's head be maintained at a level higher than the feet. It is also enhanced in the patient who is medicated to a point where he cannot cough.

Case 3—W. T., this 62 year old Negro man was admitted to the hospital on July 7, 1954, complaining of burning pain in the stomach. He had had indigestion and feeling of fullness

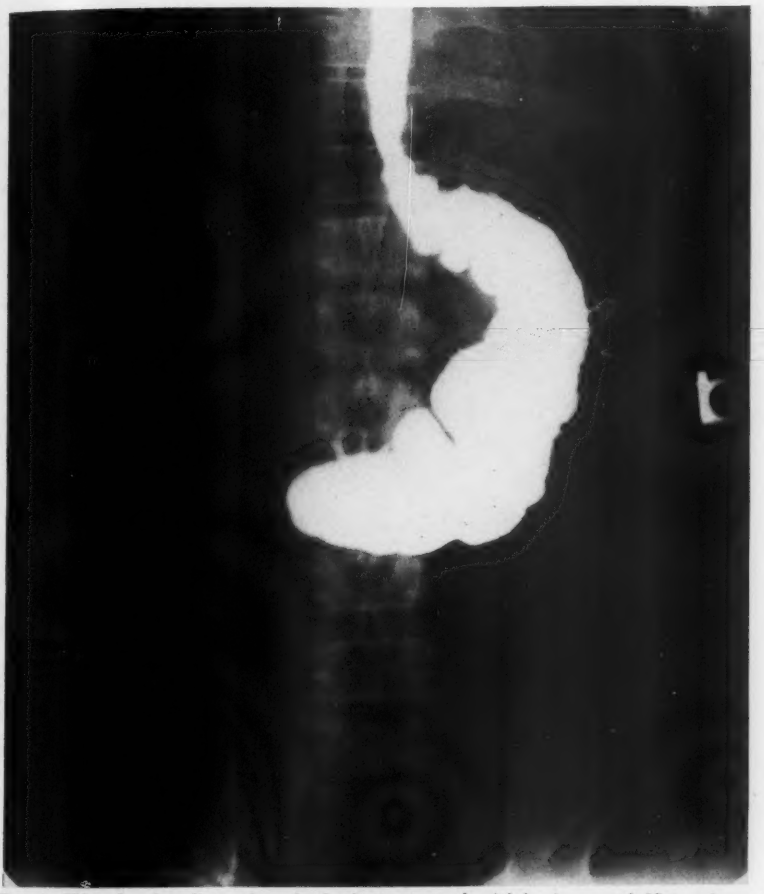

FIG. 2. Film showing distention of substitute stomach with barium meal. Note adequate esophageal anastomosis. Note delay in filling of duodenum. This is thought to be due to sectioning of the vagi (case 3).

and tightness in the epigastrium since 1931. However, about seven months prior to admission he developed more severe pains in the right upper quadrant and substernal burning. This progressed and at the time of admission he had pain occurring in the morning on awaking, relieved by eating breakfast, and returning again about 11:00 a.m., relieved by eating lunch. Just prior to admission he began to vomit at intervals, but the vomitus did not contain blood. There was a history of tarry stools once in 1953 and once in 1954 but none recently. The past history showed that he had had an appendectomy in 1926 and an inguinal herniorrhaphy in 1937. He had nocturia five or six times but no dysuria or hematuria.

Physical examination on admission showed a small, thin, Negro man, who did not appear acutely ill. He was found to have bilateral arcus senilis and arteriosclerosis of the fundi. The heart and lungs were normal. The abdomen showed the appendectomy scar. The liver was palpable 5 cm. below the costal margin but was smooth. A questionable mass was palpated in the upper abdomen extending to the left of the midline which was nontender and

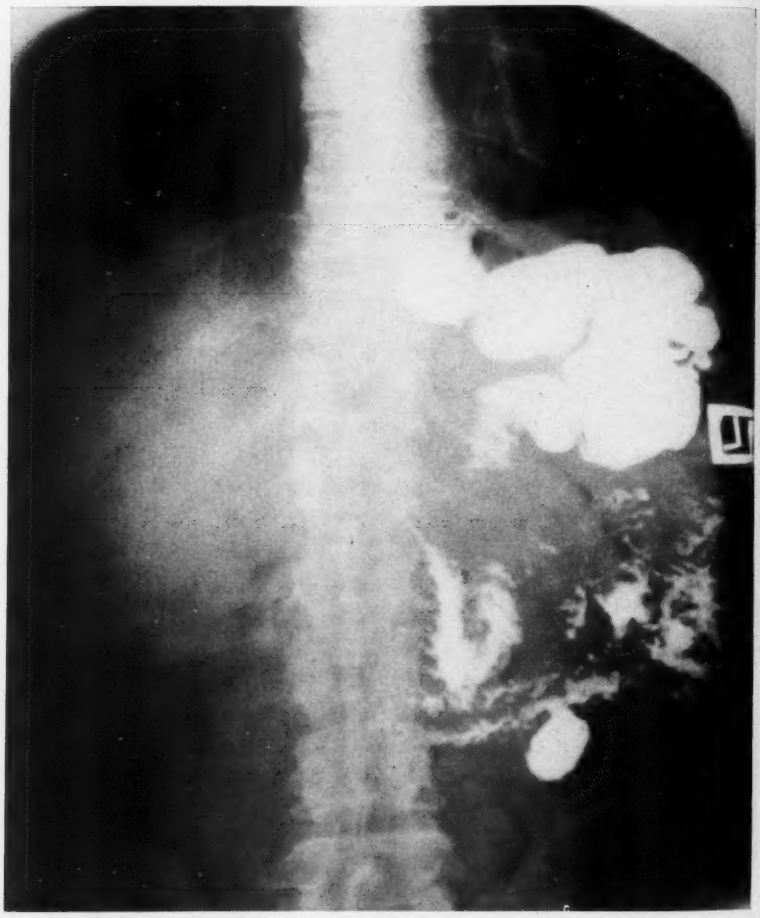

FIG. 3. Film showing distention of substitute stomach in case 1. (Barium in small bowel remains from earlier examination on same day).

not adherent. The remainder of the examination showed small internal hemorrhoids and slight hypertrophy of the prostate.

Laboratory studies on admission showed the urinalysis to be normal. The serology was negative. The white blood cells were 10,800 per cu. mm. and the hematocrit 31. Gastric analysis showed absence of free HCl. The stools were consistently positive for occult blood, 2 to 4 plus.

Chest roentgenogram on July 8 showed some enlargement of the left ventricle and calcification of the aortic knob. The impression was arteriosclerotic heart disease. Gastrointestinal studies showed an irregular filling defect in the stomach which involved the body and antrum and suggested a large fungating tumor of the stomach, either carcinoma or lymphoblastoma.

On August 2 a laparotomy was done and he was found to have a carcinoma of the distal

half of the stomach. Total gastrectomy, splenectomy and omentectomy was done. A loop of transverse colon was interposed between the esophagus and duodenum in an antiperistaltic fashion (preserving the middle colic vessel) to form a new stomach pouch. The patient's early postoperative course was relatively uneventful and he began to take fluids by mouth. On August 15 a roentgenogram of the abdomen showed a fluid level under the left diaphragm, and on that day a left subdiaphragmatic abscess was drained posteriorly. The pleural space was likewise aspirated and contained 500 cc. of sanguineous fluid. His condition improved slightly. On September 1 barium swallow showed a sinus tract extending between the two anastomotic sites. There was drainage of what appeared to be pancreatic juice from the drain to the subphrenic abscess. He vomited frequently and was unable to eat more than small feedings. Gastrointestinal studies at first showed marked slowing of gastric emptying; however, there was no retention in the 24 hour roentgenogram. The patient's condition gradually deteriorated. A Levine tube was passed through the replacement pouch and into the duodenum and he was fed through this for several days. His condition failed to improve. His course continued downhill with the development of terminal pneumonia. He died on Nov. 24, 1954, apparently as a result of malnutrition and pneumonia secondary to his operative procedure.

Comment: The unfortunate complication of abscess formation has been reported by others.[7]

SUMMARY

Three cases of total gastrectomy with transverse colon replacement are presented.

One patient is living and well. One patient died of surgical complications in three months. One patient died in the postoperative period after total resection for massive gastric bleeding unabated by subtotal gastrectomy.

The surviving patient is comfortable with no sequellae. A recent culture of the transplanted colon shows a persistence of E coli.

It is believed that when total gastrectomy is indicated, a substitute stomach may alleviate or prevent the unpleasant postgastrectomy syndrome.

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SURGICAL TECHNIC

GYNECOLOGIC OPERATIONS FOR INFERTILITY

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Surgical operations to enhance reproduction in the female have been attempted for as long as female surgery has been done. Many ingenious procedures have been described. All of the operations advocated have had one thing in common, namely, that the number of patients who have had living infants following such surgery has been extremely small. For that reason most of the surgical procedures which have been developed have had cautious reception and guarded acceptance. Ultimately most have passed into disrepute. As recently as 1937 a survey of surgery pertaining to the Fallopian tubes was reported in a most unfavorable light.[6]

With the introduction of new plastic materials, such as polyethylene, during the past few years, there has been a rebirth of enthusiasm for surgical procedures which may improve the fertility of the female. Polyethylene tubing has had its greatest role in gynecologic surgery in operative attempts to establish or improve tubal patency. A feeling of cautious optimism has been generated by some of the leading surgical specialists in infertility during the past three or four years.[8, 14] A definite improvement in results has been reported in the recent literature.[2]

Operations pertaining to the tubes, uterus, cervix, vagina and the ovaries will be considered and evaluated in light of new perspectives.

FALLOPIAN TUBES

Most of the surgical procedures which have been advocated for the correction of infertility have been concerned with the Fallopian tubes. Tubal occlusion or partial tubal obstruction may occur as the result of innumerable conditions. The most common cause of tubal obstruction always has been gonorrheal salpingitis. However, puerperal sepsis, pelvic abscesses as from a ruptured appendix, adhesions from previous pelvic operations, and surgical obliteration of the tubes from a previous ectopic pregnancy, or by means of tubal ligation, all may be contributing factors. Unfortunately, attempts at demonstrating tubal patency in previously normal tubes have at times resulted in subsequent tubal occlusion due either to the type of material used or due to the introduction of infection.

Before any surgical procedure on the Fallopian tubes is contemplated several prerequisites must be met.[3] The fertility of the husband should be established beyond doubt. That the patient is ovulating should be verified by basal temperature readings and by endometrial biopsy. No incompatibility should exist between the sperm of the husband and the cervical mucus of the patient. The patient and her husband should be fully informed and completely aware of the fact that there

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still is a rather high percentage of failure in all tubal surgery. That tubal surgery may be beneficial should be established without equivocation.

There are several ways of demonstrating tubal patency or impatency. At the present time the instillation of normal saline solution through the tubes with or without the addition of phenolsulfonphthalein is being used. If there is any doubt as to the patency of the tubes when tested with the saline solution it is imperative that an hysterosalpingogram be done. The hysterosalpingogram is the only method of showing the exact site of tubal obstruction.[9] If the patient has a low pain threshold or if she is extremely apprehensive the hysterosalpingogram is best done under selfadministered trilene analgesia or under pentothal anesthesia. The analgesia or anesthesia serves the valuable purpose of eliminating tubal spasm. It is important not to use any contrast material which may cause tubal damage. Salpix or Skiodan-Acacia are being used at the present time. The use of Lipiodol and other oily contrast media has been discontinued. Tubal occlusion secondary to oil granulomas, acute peritonitis, and oil emboli have occurred when iodized oil has been used as the contrast medium.

The exact importance of the fimbria of the tube has not been demonstrated positively, but clinical experience has shown that patients with damaged fimbria have a greatly reduced chance for conception even though the proximal portion of the tube is patent. Patients who have no tubal patency demonstrable by hysterosalpingogram should be given little hope for successful tuboplasty at this time, although occasionally the impatent portion in the medial portion of the tube can be resected and the distal part of the tube then anastomosed to the uterus.[7]

When the decision has been reached to operate upon the patient with occluded tubes the patient first is placed in the lithotomy position. Ten cc. of methylene blue then is injected into the endometrial cavity by means of the Rubin cannula. The patient then is placed in the supine position and the abdomen opened. The pelvic viscera are thoroughly explored first. By means of the methylene blue one can determine the exact extent of tubal patency or impatency. If pelvic adhesions are present, lysis of those adhesions is done. The fimbriated ends of the tubes must be completely mobilized. Fine polyethylene tubing then is introduced through the fimbriated end of the tube or through the farthest point in the tube where patency has been demonstrated by the dye. Whenever possible the polyethylene catheter should be passed through the tubal lumen into the uterine cavity. In the event that that is impossible, the obstructed portion of the tube should be excised and the distal portion of the tube anastomosed to the endometrial cavity with the polyethylene tubing in place. The free end of the polyethylene tubing in the abdomen then is sutured to the serosa of the tube by fine absorbable catgut sutures. The catheter or catheters then are brought through the cervix from above if the endometrial cavity has been opened or from below if one has been successful in passing the polyethylene tubing into the uterus through the tubal lumen. The catheter or catheters then are sutured to the cervix. The tubing should be left in place for approximately six weeks.

Recently Rock has been using a polyethylene *tent* over the freed up fimbriated end of the tube to prevent postoperative adhesions and readherence of the tube

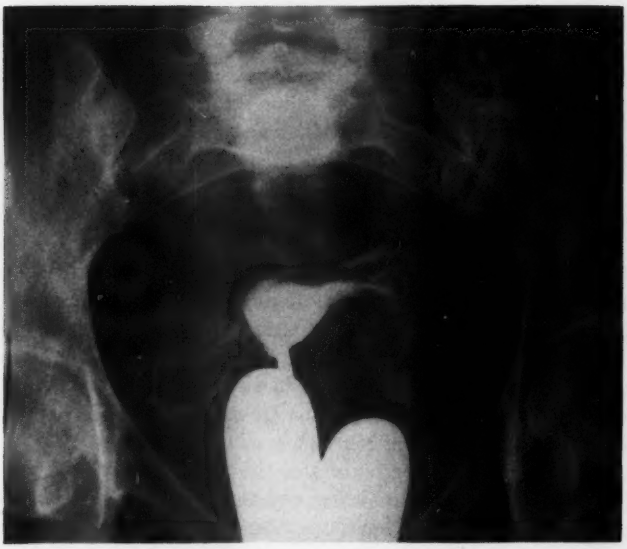

FIG. 1: D. G. A tubal ligation had been done elsewhere in 1947, under the mistaken impression that the patient had tuberculous retinitis. A correct diagnosis of Brucellosis subsequently was confirmed, and the condition had been successfully treated with antibiotics. The above salpingogram was done on Sept. 19, 1953 with Visco-rayopake. Both tubes were closed. Although excessive pressure was not used the radiopaque material entered the uterine veins. A bilateral tuboplasty was done on Oct. 20, 1953 over polyethylene catheters. The polyethylene was removed six weeks later. L.M.P. Dec. 22, 1953. Patient was delivered of a 9 lb. 7 oz. girl infant on Sept. 23, 1954. The patient again is pregnant (1955).

to its surrounding structures.[14] At the present time that procedure necessitates a second laparotomy within a few weeks for the removal of the polyethylene. It recently has been suggested that additional polyethylene tubing be attached to the tent and that portion of the tubing brought out through the culdesac into the vagina at the time of the primary procedure. By means of a posterior colpotomy it then would be possible to remove the tent or tents from the fimbriated end or ends of the tubes without subjecting the patient to a second laparotomy.

In patients who have had a previous tubal ligation, anastomosis of the tubes after resection of the obstructed area is best done over polyethylene catheters. It would appear that the patient who has had a simple tubal ligation in the medial aspect of the tubes and in whom the fimbriated ends are perfectly normal should have the most favorable results from a tuboplastic procedure (fig. 1).

The after care of such patients is important. Following removal of the plastic tubing saline solution should be injected through the tubes after each menstrual period for several months to insure a continuation of tubal patency.

In the patient who has an ectopic pregnancy, ruptured or unruptured, an attempt should be made to preserve the function of the tube, if the condition of the patient permits. There should be no place in good gynecologic surgery for

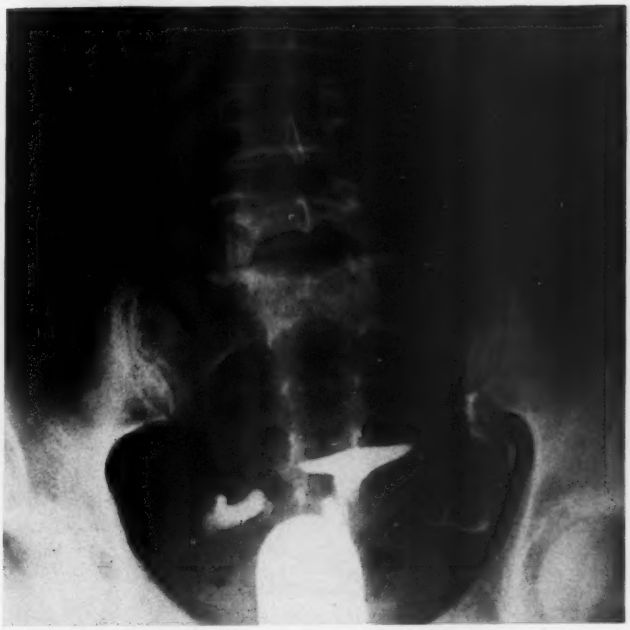

FIG. 2: P. B. An unruptured ectopic gestation of six weeks duration was resected from the distal third of the left tube on Jan. 25, 1954. The tube was sutured open instead of being clamped, cut and closed. Salpingogram done on Feb. 26, 1954 showed both tubes to be open.

needless clamping, cutting and the closure of the tube by a suture ligature in the presence of an ectopic gestation. In unruptured tubal pregnancies it always is possible to make an effort to maintain tubal patency. If the ectopic gestation is at the fimbriated end of the tube an attempt should be made to maintain patency by suturing open that portion of the tube (fig. 2 and fig. 3). The polyethylene tent of Rock might well have a place in this situation. With an ectopic gestation in the middle third of the tube anastomosis over a polyethylene catheter (if the condition of the patient permits) is an extremely valuable procedure when continued fertility is desired by the patient. If the pregnancy has been in the cornual portion of the tube, and rupture has occurred, the condition of the patient generally will not permit unnecessary and time consuming surgery. Under favorable circumstances, however, it is conceivable that the distal portion of the tube might be reimplanted into the endometrial cavity with a polyethylene catheter in place.

UTERUS

Surgical procedures on the uterus for infertility include endobiopsy which is done on the first day of the patient's menstrual period. That is used to establish the presence or absence of ovulation during that particular cycle.

An operation on the uterus which has a definite but extremely limited place is

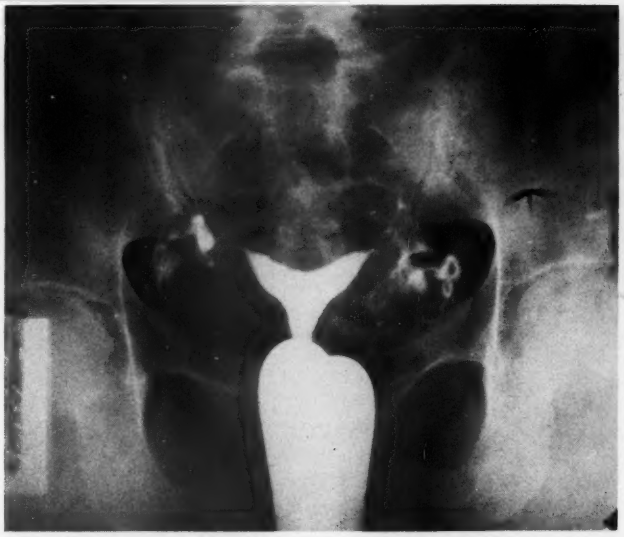

FIG. 3: S. L. An early ectopic gestation was removed from the outer third of the left tube in July 1954. The above salpingogram was done on Sept. 10, 1954. Both tubes were found to be patent. (Patient of Dr. Wm. McKelway).

the unification operation of Strassmann for correction of the anomalous uterus didelphys.[17] That is used to best advantage in the patient who has a complete failure of fusion of the Muellerian ducts. Infertility and abortion are common in such women. The unification operation as outlined by Strassmann consists in cutting the cervical part of the septum from below and then abdominally opening the uterus and excising the septum between the two halves of the uterus. It is a valuable procedure in women with anomalous uteri who have had infertility or habitual abortion problems.

Myomectomy for fibroid tumors of the uterus is of value in many patients who have had prolonged infertility. The tumor should be of fairly large size, it should be distorting the endometrial cavity or it should be impinging upon the cornual portions of the tubes before myomectomy is considered.[13] Small myomas in general rarely cause infertility. The gynecologist who proposes myomectomy should be aware of the possibility that an hysterectomy is at times the eventual outcome of the operation even when myomectomy had been thought to be possible of attainment.

Uterine suspension, although advocated by some authors[5] for the improvement of fertility, is a surgical procedure that has been greatly maligned by its too frequent employment. Only in patients who have had long standing infertility problems where all other factors have been corrected or eliminated should suspension of the uterus ever be considered. Certainly no uterus should ever be suspended until after an adequate trial with a pessary has been completed. The operation is of little proved value for the enhancement of reproduction.[3] It

seldom is indicated and rarely successful. It is one of the most frequently employed procedures in women who have subsequently been shown to have hysteria.[4]

CERVIX

All cervical lacerations incurred at the time of delivery should be repaired immediately. Such repairs are uniformly more successful than are repairs of the cervix done after the puerperium. Cervices in which lacerations have not been repaired may cause difficulty in subsequent years due to cervical incompetency.

A recent procedure advocated by Shirodkar of Bombay appears to be extremely valuable in the correction of cervical incompetency.[15] Its particular application is in the patient who has had repeated abortions during the second trimester of gestation. Those patients will be found to have an incompetent cervix which is unable to contain the enlarging products of conception. Ultimately the cervix effaces and dilates allowing the fetal membranes to prolapse and then rupture with inevitable interruption of the pregnancy. The procedure consists of anterior and posterior incisions through the mucosa of the cervix. A purse string ligature of fascia lata taken from the thigh then is placed under the mucosa and around

FIG. 4: E. M. Nulligravid after five years of marriage. The patient had absolute failure of fusion of the Muellerian ducts with complete vaginal septum as shown above. After all causes for the prolonged infertility had been eliminated, surgery was performed. The septum was excised from below. A unification operation on the uterus then was done abdominally. Conception has not occurred as yet.

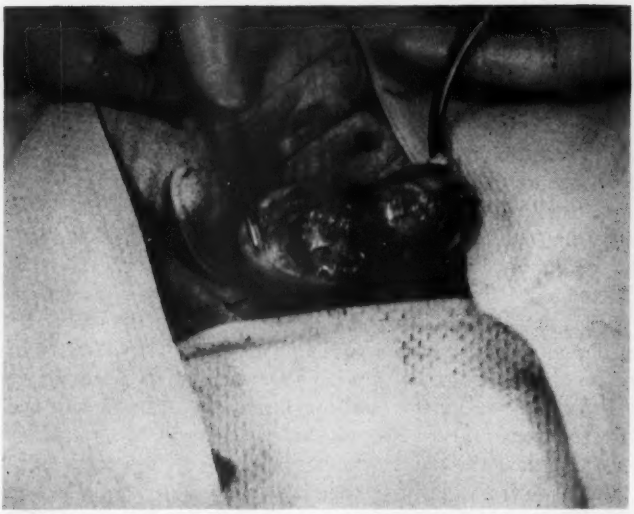

FIG. 5: B. L. 16 years old. This patient had bilateral dermoid tumors of the ovaries. The left ovary measured 25 by 25 by 15 cm. and had to be removed completely. The small dermoid cyst in the remaining ovary then was exposed as shown above. (Patient of Dr. James Sites).

the cervix, tightened, and then sutured into place at the level of the internal os. The procedure may be done during gestation which gives it a distinct advantage over the procedure advocated by Lash.[10] The results of the Shirodkar procedure have been quite encouraging.

VAGINA

Vaginal surgery for infertility is concerned primarily with the removal of the vaginal septum in patients who have either a complete or an incomplete septum of the vagina. That procedure is easily carried out. The removal of a complete septum should theoretically improve the patient's chances for conception (fig. 4).

Incisional surgery of the hymenal ring is unnecessary except in extremely rare instances. Simple dilatation of the hymenal ring can be done easily by the patient using a dilator. The operation of *hymenotomy* has outlived its usefulness as a gynecologic procedure. Hymenal incisions have caused far more dyspareunia than they have eliminated. It is difficult to see how the addition of a painful new incision in the introitus can do anything but add to the problems of a patient who already has painful sexual relations.

OVARIES

Ovarian surgery for the correction of infertility generally is limited to ovaries enlarged by tumors, by endometriomas, or by the *pale white ovaries* of the Stein-Leventhal syndrome.

When surgery has to be done for a dermoid cyst in women in the childbearing

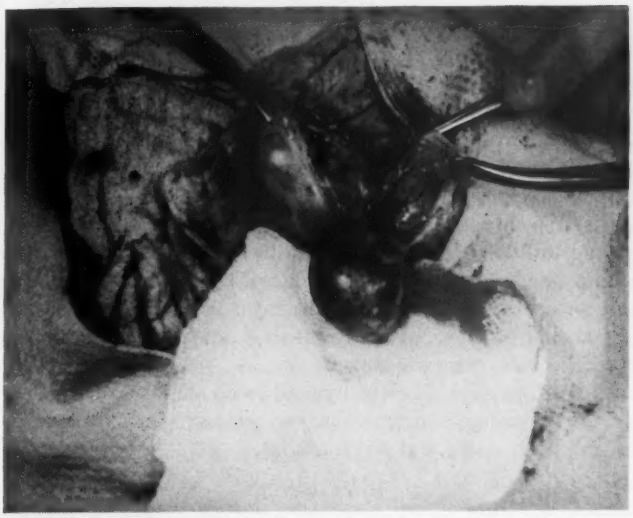

FIG. 6: Same patient as figure 5. Photograph shows the ovary at the time of resection of the dermoid tumor from the central portion of the ovary. The two halves of the ovary then were reunited conserving normal ovarian function. Castration which might have resulted was prevented.

age an attempt should be made to resect the tumor rather than to remove the ovary (figs. 5 & 6). Sacrifice of an entire ovary instead of a partial resection requires neither surgical judgment nor surgical skill. Ovarian resection, conversely, requires delicate gynecologic technic. Needless removal of the entire ovary in a few minutes time may adversely affect the patient for the rest of her childbearing life.

Endometriosis of the ovaries is fairly frequent in infertility patients. Ovarian resection can practically always be done satisfactorily with such lesions. In most instances there will be an improvement in the fertility index of the patient.[11] Any infertile patient who has a persistent ovarian tumor or tumors should be explored after a reasonable period of observation.[12] Cystic tumors may be observed for longer periods of time than solid tumors because of the higher incidence of carcinoma in the latter type. Obviously any rapidly enlarging ovarian tumor or any ovarian tumor causing pain should be investigated without delay.

With the Stein-Leventhal syndrome which consists of bilaterally enlarged, hard, white ovaries, bilateral ovarian resection will occasionally result in the resumption of ovulation.[16] Prior to surgery, anovulatory bleeding is the rule. The actual improvement in fertility after the operation, however, has been disappointing.[1]

Conservatism in all ovarian surgery in women in the childbearing age will do much to prevent later infertility problems. Similarly conservation of the tube often can be accomplished even though an ovary may require complete surgical excision.

SUMMARY

Enthusiasm for tubal surgery in the correction of infertility has recently been given a new impetus by the introduction of polyethylene.

Whenever possible tubal patency should be conserved when surgery is done for ectopic pregnancy.

The unification operation has a definite place in women with anomalous uteri who have infertility problems or habitual abortions.

Uterine suspensions have little place in the gynecologic surgery of infertility.

A recent successful operation for cervical incompetency has been described.

Conservatism should be the hallmark of all ovarian surgery in girls, young women and women still in the childbearing age.

No ovary should be removed in a young woman for a benign condition when ovarian resection can be accomplished.

Cautious optimism should be the byword in all surgery for the improvement of fertility. An extravagant promise by the gynecologist may lead to ultimate heartbreak for the patient and her husband.

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STAGE OCCLUSION AND RESECTION OF THE HUMAN AORTIC ARCH WITH HYPOTHERMIA

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The challenge of grafting the entire aortic arch is being studied in the laboratory by a variety of methods including shunts, hypothermia, and extra-corporeal circulation technics. Successes in the experimental laboratory now are being applied to human beings. Schafer and Hardin,[3] using polyethylene shunts, succeeded in grafting a human arch in 1952. The patient died one hour later from ventricular fibrillation. More recently Stranahan, and associates[4] have used temporary homologous or heterologous vessel grafts to bridge the occluded segment during grafting. This affords an expansile large bore shunt, but adds to the operative time in an already lengthy procedure. They reported a case of a human being in whom this method was successful, but the patient died in the anesthetic recovery period as a result of hemorrhage from the left pulmonary artery stump. An incidental pneumonectomy had been done.

Hypothermia is recognized now as a useful adjunct in large vessel grafting to protect vital tissues during the circulatory occlusion period.[1, 2] Laboratory experiments combining hypothermia with shunting technics in our experience have been quite encouraging.

This report is to record the use of hypothermia combined with staged segmental occlusion in resecting an aortic arch aneurysm in one human being. The latter technic is a natural outgrowth of our experience with shunts. It differs from the work of Stranahan and associates[4] in that the segmental shunt remains as a permanent feature of the reconstructed arch. To our knowledge this is the first attempt in a human being of either technic.

CASE REPORT

A 48 year old Negro man was admitted to the George Washington University Hospital on June 17, 1954, complaining of weakness. Seven days before he noticed giddiness, nausea, and promptly fell to the floor. He became unconscious and remained so for 5 to 7 minutes. After his recovery he was able to continue normal activity. Two days before admission he again felt dizzy, lost his balance and fell. He did not lose consciousness but developed left-sided weakness and could not stand without help. He had generalized weakness, loss of strength in his left arm, and difficulty in swallowing up to the time of admission. There was no history of hypertension. At one time he was treated for syphilis.

On physical examination he was a well developed, muscular, middle aged Negro man in no distress. The left arm was flaccid, and he exhibited a speech impairment. He was alert and cooperative.

The radial and carotid artery pulsations were faintly perceptible. The pulsations of the vessels of the lower extremities were excellent. The blood pressure in the upper extremities

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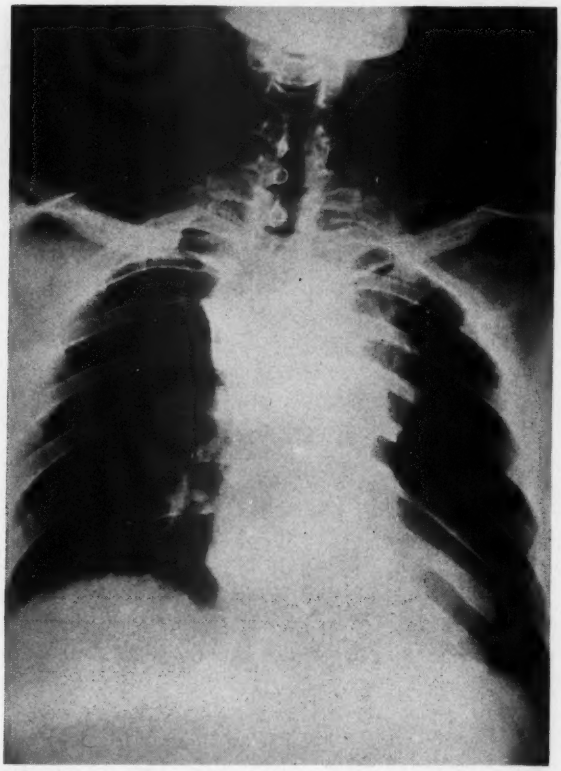

FIG. 1. Preoperative roentgenogram of the chest showing large calcified mass in the superior mediastinum (aneurysm).

was approximately 76/70 and in the lower extremities 170/90 on the right and 200/90 on the left. Left facial muscular weakness was observed. The lungs were clear. The heart was not enlarged, and no abnormal sounds were heard. There was motor weakness combined with hyperactive reflexes in the left arm.

The laboratory reported the hematocrit 49 per cent, hemoglobin 14.4 grams, leukocyte count 10,400 per cu. mm. with 70 per cent neutrophils, 34 per cent lymphocytes, and 4 per cent monocytes. The urine concentrated to 1.024 and showed a trace of albumin. The cardiolipin (V.D.R.L.) and Kahn serology studies were negative for syphilis. Coagulation time by the Lee White method was 8 and 9 minutes, and the prothrombin time was 64 per cent of normal. An electrocardiogram was reported as showing left ventricular hypertrophy with myocardial ischemia.

Roentgenographic studies of the chest proved the left ventricle of the heart to be enlarged, the lung fields normal, the trachea deviated to the right, and a large uniform calcific mass in the superior mediastinum (fig. 1). This mass could not be separated from the arch and the first portion of the descending aortic arch. On barium swallow the esophagus was displaced posteriorly and to the right of the mass. An angiocardiogram was attempted. The innominate vein was outlined, but dye was not seen in the cardiac chambers or the mass.

The impression was that there was an aneurysm of the arch of the aorta. Exploration was

recommended, but the patient refused to be operated upon and left the hospital on the tenth day.

He was readmitted to the George Washington University Hospital on Sept. 23, 1954 requesting an operation because of the downhill course since his last admission. In the interval, he had continued to have vertigo, weakness, and fatigability. Mental concentration had become poor.

On physical examination the patient was a well developed, but poorly nourished 48 year old man, who appeared chronically ill. Mental deterioration since his last admission was obvious. The carotid and subclavian artery pulsations were absent. The blood pressure could not be obtained in either upper extremity. The blood pressure in the right lower extremity was 160/85. The left arm was stronger than the right, and his facial weakness had disappeared. A systolic murmur, grade I, could be heard over the upper thoracic vertebra and second left interspace anteriorly.

The laboratory reported hemoglobin 17.7 grams, hematocrit 44 per cent, leukocyte count 8,500 per cu. mm., 79 per cent segmented leukocytes, and 21 per cent lymphocytes. The urine concentrated to 1.022. Blood urea nitrogen was 9 mg. per cent, serum chlorides 99.8 milliequivalents per liter (mEq/L.), serum CO_2 combining power 22 mEq/L., serum sodium 148 mEq/L. and the total proteins 7.25 grams per cent with an albumin-globulin ratio of 2 to 1. The chlorides were elevated to 103 mEq/L. on the fourth hospital day.

Roentgenographic studies were unchanged since the last admission. The electrocardiograms indicated left ventricular hypertrophy.

The strength of his left arm returned. The mental deterioration remained unchanged.

OPERATION

On September 4, the patient was placed on a mattress for cooling. Satisfactory anesthesia was maintained through an endotracheal tube utilizing ether, nitrous oxide, and oxygen. The mattress for cooling was filled with iced water (15 C.) and maintained by continuous flow. A transsternal incision was made entering both pleural cavities through the third intercostal spaces. The right clavicle and costal cartilages of the first, second, and third ribs were divided and retracted for a more adequate exposure.

FIG. 2. First stage of the reconstruction. (Homologous graft anastomosed to the ascending aorta).

FIG. 3. First stage of the reconstruction completed between the innominate artery and ascending aorta (note aneurysm in background).

A large aneurysm involving the entire aortic arch, medial side of the innominate, left carotid, and medial side of the left subclavian arteries was dissected from the surrounding tissues. There were no pulsations of the distal innominate, carotid, and subclavian arteries. The distal segments of the innominate and left subclavian arteries appeared normal. The left carotid artery appeared cord-like. The ascending and descending portions of the aorta were normal in appearance except for slight dilatation. The trachea and esophagus were displaced to the right by the aneurysm.

The rectal temperature was 30 C. The left innominate vein was divided and ligated. Umbilical tapes were placed around the proximal and distal aorta and its tributaries. A Beck clamp was applied to the superior border of the ascending aorta without obstructing the blood flow. A reconstituted freeze-dried homologous graft of the descending thoracic aorta was used to provide a shunt between the ascending aorta and the distal innominate artery. This was anastomosed to the ascending aorta (fig. 2). A similar clamp was applied to the inferior lateral border of the innominate artery at its bifurcation and the distal end of the graft was anastomosed to the innominate (fig. 3). The graft was aspirated of air and filled with saline solution. The clamps were released and immediate pulsations in the right carotid and subclavian arteries were noted.

The innominate artery was cross-clamped between the graft and aneurysm and divided. The lumen of the innominate artery was almost completely occluded at this point by thrombus and atheromatous plaques. The vessel wall was so friable that repeated suturing was necessary to control bleeding.

The left carotid artery was found on exploration to be totally occluded by organized clot and atheromatous plaques. It was divided between ligatures. The left subclavian artery carried a minimal quantity of blood. It was clamped with 2 Pott's ductus clamps and divided (fig. 4). The rectal temperature was 27.7 C.

The aneurysm was clamped at its base and excised. Atheromatous plaques and arteriosclerotic degeneration with thrombus formation involved approximately ⅔ of the circumference of the aorta. A lateral aortorrhaphy was attempted but abandoned as there was insufficient vessel wall for suturing. The ascending aorta was cross-clamped distal to the first

FIG. 4. Division of the aneurysm from the three branches of the aortic arch (aneurysm retracted downwards).

graft, and the descending aorta was clamped at the ligamentum arteriosus. The arch, including the base of the aneurysm, was excised between these clamps. A reconstituted freeze-dried homologous aortic graft was used to restore the excised segment. The innominate, left carotid, and left subclavian arteries of the graft were cross-clamped using Pott's ductus clamps. The occluding clamps on the aorta were released. Excellent blood flow was observed through the graft. The time of complete occlusion was 23 minutes.

The innominate and left carotid arteries of the graft being redundant, were sutured at their bases and excised. A homologous thoracic aorta graft was tailored to bridge the gap between the graft and host subclavian arteries. After anastomosis the clamps were released and a good pulsation was observed in the subclavian artery. This could be felt peripherally (fig. 5).

The blood pressure as measured in the right arm remained approximately 80/40 throughout the entire procedure. The body temperature remained at 27 C. during this reconstruction period. The patient was warmed by passing warm water through the mattress, and by a continuous flow of warm saline solution (45 C.) perfused into the pleural cavities. When the rectal temperature reached 35 C. the chest wall was closed, leaving a catheter in each pleural cavity connected to water-sealed drainage bottles.

Pathology: Two specimens were received labeled aneurysm and the aortic arch. The aneurysm measured externally 12.5 by 10 by 5.5 centimeters and internally 6 by 4 by 4 centimeters. The posterior wall consisted of a thick, friable, brownish, organized clot. The anterior wall measured 0.33 centimeters in thickness and numerous atheromatous plaques were noted. The lumen of the aneurysm contained an organized clot. The innominate and left subclavian vessels were closely adherent to the aneurysm, forming part of its wall. The left carotid extended from its superior surface. The wall of these vessels was thickened and contained atheromatous plaques. The aortic arch was 10 centimeters in length. There was a defect on the superior border of the aortic arch which extended $\frac{2}{3}$ of the circumference of the vessel. The edges of the defect were thickened and contained numerous atheromatous plaques.

On microscopic examination the aneurysmal wall contained connective tissue, calcium,

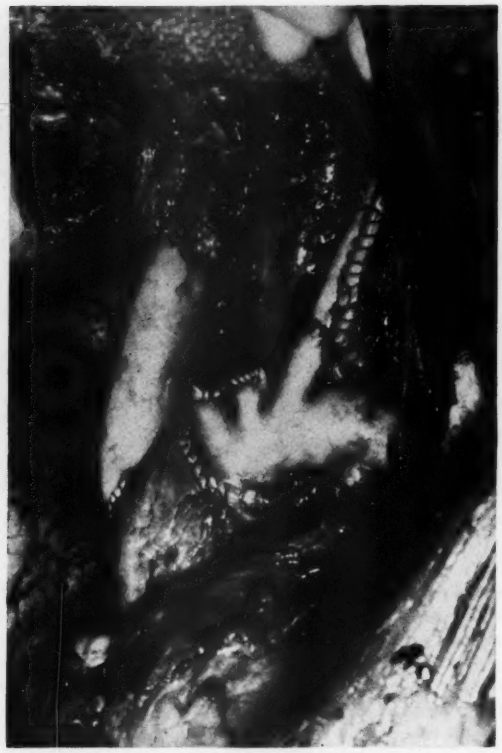

FIG. 5. Completion of reconstruction of the entire aortic arch

and laminated clot. The wall of the aortic arch adjacent to the aneurysm was composed of adventitia only which was partly replaced by connective and fibrous tissue. Lymphocytic perivascular infiltration was present in the adventitia. Final diagnosis: syphilitic aneurysm of the aortic arch.

Immediately after returning to the ward, the blood pressure was 80/55 in the right arm, pulse 64, and temperature 37 C. He answered questions and complained of pain. A continuous intravenous infusion of 1500 cc. of 5 per cent glucose in distilled water containing 30 mg. of neosynephrin was given to maintain the blood pressure during the following 12 hours. The blood pressure remained around 80/60, and pulse varied from 72 to 120 per minute. The patient was oriented and could answer questions. There was no evidence of central nervous system damage, and he could move all extremities.

Laboratory studies during his postoperative course showed a hematocrit 47 per cent, hemoglobin 14.4 grams per cent and leukocyte count 8,750 per cu. mm. The blood chemistries showed serum chlorides 97.8 mEq/L., CO_2 combining power 12 mEq/L., serum sodium 144 mEq/L., and blood urea nitrogen 24 mg. per cent.

The patient's condition remained unchanged for 12 hours. Urinary output at this time was 25 cc. A transfusion of 500 cc. of whole blood was given.

At 9 o'clock the following morning the blood pressure and pulse suddenly were unobtainable in either arm. Respirations ceased. The patient did not respond, and in spite of all therapeutic measures, could not be revived. He was pronounced dead 12 hours and 35

minutes after completion of the surgical procedure and 15 hours following resection of the aortic arch with replacement by homologous grafts.

DISCUSSION

Despite exhaustive efforts, postmortem examination permission was denied. For this reason certain aspects must go unanswered. The clinical and laboratory data remain as the sole support in reconstruction of the postoperative period.

The collateral circulation undoubtedly is a unique factor. The progression of the disease was slow enough to allow a collateral blood supply through the intercostal vessels to reach the cerebral circulation.

Staged occlusion of the blood flow was a second major factor. The first stage consisted of partial occlusion of the ascending aorta, thereby establishing a homologous graft shunt between the arch and the innominate artery. It then became a permanent part of the reconstruction allowing cross-clamp occlusion distal to the anastomosis. The remainder of the arch then could be reconstructed by two additional homologous vessel grafts. At no time was there a complete interruption of arterial blood flow to the cerebrum, in fact, once the first stage was accomplished, cerebral flow was markedly improved. This was substantiated by the development of pulsations in previously pulseless right carotid and subclavian vessels.

Hypothermia served as the third important factor in protecting vital tissues during partial and complete stages of occlusion. The correlation of body temperature, serum sodium and potassium levels are presented in table I. Blood

TABLE I

Hour	Temperature Degrees Centigrade	Serum Sodium Milliequivalents Per Liter	Serum Potassium Milliequivalents Per Liter
AM			
8:30	Anesthesia started		
9:00	Operation started—cooling through mattress begun		
9:00	38		
10:00	37	145	3.9
11:00	34.8	137	3.6
12:00	33	138	3.4
PM			
1:00	31	149	3.6
2:00	30	144	3.6
3:00	29	127	4.1
4:00	28.5	144	4.2
5:00	28	140	3.2
6:00	27.5	133	3.0
7:00	Warming begun, mattress alone		
7:00	27.2	102*	6.8*
8:00	28.8	157	3.5
9:00	Warming begun, intrathoracic		
9:00	31.5	157	3.4
9:30	34		

* Error due to technical difficulties.

electrolytic changes were influenced by transfusions of stored whole blood (6,000 cc.) during the procedure. The significance of electrolytic changes under hypothermia is not fully understood.

The combination of the above three factors served to protect this patient from additional cerebral damage, spinal cord anoxia and renal failure.

Continuous electrocardiographic tracings studied throughout the procedure did not indicate any period of myocardial anoxia or irregularity. This was true during induction of hypothermia as well as during the staged occlusions.

The sudden death of the patient is thought to be of an embolic nature. Acute circulatory collapse in such cases usually is due to coronary or pulmonary embolism. Hemorrhage was not evident in the chest drainage tubes in either hemithorax. Tracheal aspirations did not reveal obstructive secretions.

The duration and extent of such a procedure implies considerable stress on multiple physiologic processes. Adrenal exhaustion may have played an important role during operation as well as afterwards. Replacement therapy was not given.

SUMMARY

A case of syphilitic arterial aneurysm involving the entire aortic arch is reported.

The successful reconstruction of the entire arch after excision of the aneurysm is described.

To our knowledge this is the third such attempt to reconstruct the entire aortic arch in the human being.

The technics of staged occlusion, utilizing permanent homologous vessel shunts and hypothermia, are presented for the first time.

A brief review of surgical technics in aortic arch reconstruction is given.

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TOTAL MAMMARY GLAND EXCISION WITH IMMEDIATE BREAST RECONSTRUCTION

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Total removal of the mammary gland is indicated occasionally in the management of advanced hyperplasias and hypertrophies with diffuse and widespread fibrocystic disease. Complete excision of the gland may be required in the case of large benign tumors; it may be mandatory in the resection of similar growths known to have a predisposition to malignant change as in cystosarcoma phyllodes.

Simple mastectomy, as routinely done, results in unsightly scars, a flat chest, and absence of the areola and nipple. Esthetically the operation constitutes a highly disfiguring procedure. Psychologically, the surgical result offers an ever present source of mental trauma. In all probability simple mammectomy has been justly condemned as a therapeutic measure too radical for management of benign lesions of the breast.[7]

Excision en bloc of all glandular tissue and the creation of a completely acceptable breast facsimile may be accomplished in a single operative procedure. Following amputation of the mammary gland reconstruction may be achieved by a regional dermo-fat flap and free nipple graft, by skin-adipose tissue flaps, or by a free dermo-fat-fascia transplant. The choice of procedure is based upon the relative amounts of fatty and glandular tissues comprising the subcuticular composition of a particular breast.

The history of mammoplastic procedures is concerned primarily with methods designed for the correction of hypertrophy and ptosis. There is infrequent mention of repair subsequent to simple mammectomy. Free fat grafts have been implanted subcutaneously to fill defects resulting from surgical ablation. For example, in 1895, Czeany corrected a breast defect with a lipoma obtained from the patient's thigh. The contour temporarily was good; finally, absorption of the fatty tumor left only an unsatisfactory scar.[9] Lexer removed the total gland for chronic cystic mastitis through a thoracomammary incision. He immediately reconstructed the breast by rotating fatty axillary tissue into position beneath the preserved skin and nipple. Lexer was well aware of the absorption following such a transplant.[11] Rosenour in 1951 used free fat transplants to restore breast contour; this procedure, too, was followed by considerable absorption.[18]

In 1950 Maliniac first suggested that the flatness resulting from total excision of the mammary gland could be permanently corrected by the insertion of a "posterior de-epithelized flap with its attached adipose tissue" beneath an anterior flap. The superficial upper flap was delineated by a concave lunar incision

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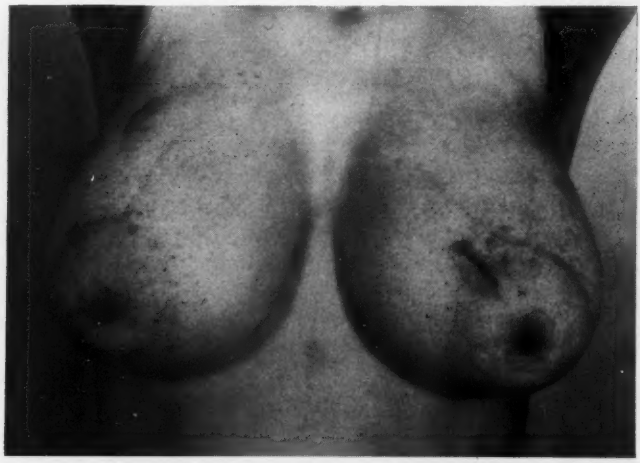

FIG. 1a

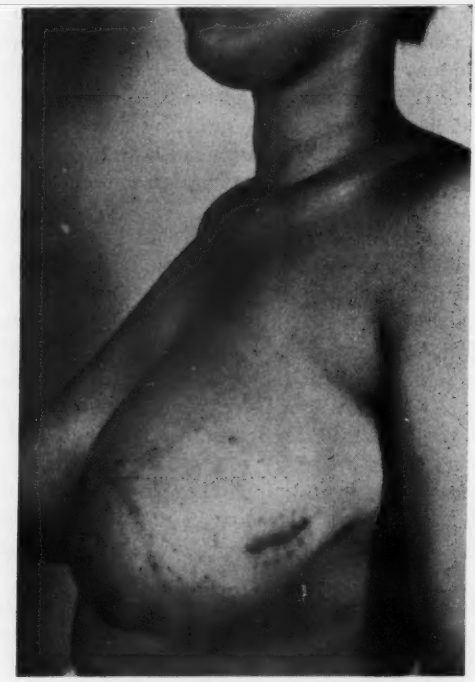

FIG. 1b

FIG. 1. a) Nineteen year old patient with multiple fibroadenomata and widespread fibrocystic mastopathy. Scars are result of previous excision biopsy sections. b) Side view shows additional biopsy site.

reaching in its lowest portion to a point above the superior margin of the areola. The "de-epithelized" lower posterior flap reached from the inferior areolar margin to just above the submammary fold. After transposition of the flaps a free nipple graft was sutured in position.[14]

Some three years later Longacre reported the use of a dermofat pedicle or pedicles fashioned from the lower segments of the breast to recreate normal contour after resection. These pedicles were based superiorly and rotated as much as 135 degrees; the top of the flap was fixed to the pectoral fascia in the region of the third rib.[12]

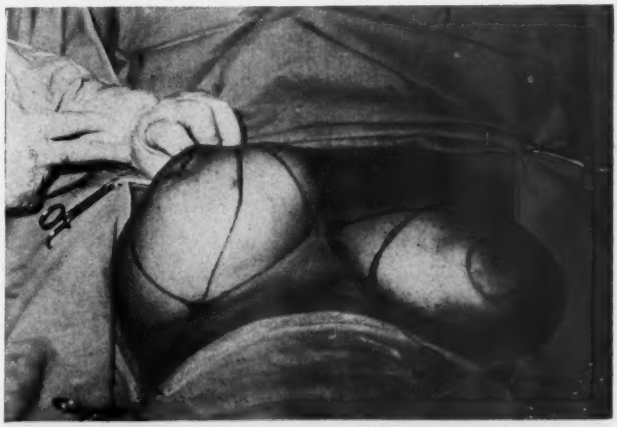

FIG. 2. Skin markings show inframammary crease and parallel line on the anterior breast surface. Superior skin-fat flap and inferior lipo-dermal flap are delineated. The nipple-areolar graft is outlined on left breast. Future nipple site is indicated also on left breast by "x"; it is in midclavicular line 21.5 cm. below clavicle. Point of intersection of midclavicular and inframammary lines is shown on right breast.

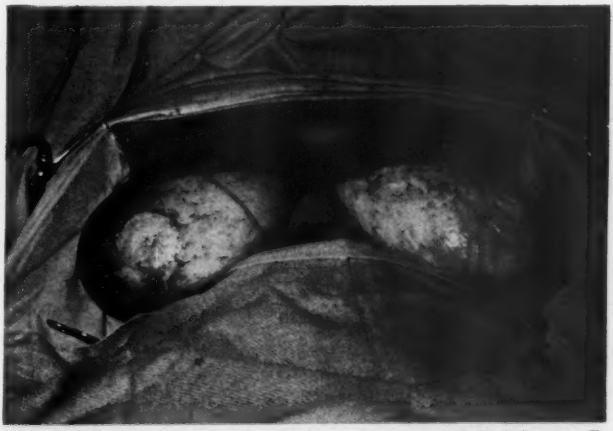

FIG. 3. Right breast: Areolar-nipple graft has been removed. Left breast: Derma covers inferior portion of breast after removal of thick split skin graft.

It is the purpose of this paper to emphasize that the restoration of acceptable breast contour is compatible with total en bloc excision of the glandular elements. There is no one reconstructive method of choice; the procedure employed depends upon the composition of the original breast. The method preferred by the authors for the average breast utilizes a lipo-dermal pedicle flap; this procedure is described in detail. Reconstruction concomitant with massive fatty hypertrophy is illustrated. Restoration in the thin undersized breast is very briefly described and diagrammed. Presurgical planning and analysis of the component portions of the breast are essential in all cases.

The breast composed originally of an appreciable quantity of fatty subcutaneous tissue and an average amount of glandular element deserves first considera-

FIG. 4. Right breast: Subcutaneous simple mastectomy. Glandular elements surrounded by fat. Left breast: Superior flap sutured in position over deeper lipodermal pedicle flap.

FIG. 5. Right breast: Upper and lower flaps are shown after glandular portion of breast has been completely removed.

tion (fig. 1). This problem presents itself most frequently; its solution is more complex. The operation combines basic surgical and esthetic principles into a one stage operation; total excision of the mammary gland and restoration of a reasonable breast facsimile is simultaneously accomplished. Excision of the gland is easily done through incisions designed primarily for the reconstructive procedure. The repair presupposes a thorough knowledge of the basic mammoplasty operations.[2, 8, 14, 15]

TECHNIC

The contemplated incisions and regional flaps are carefully outlined using any of the several skin marking materials. A line is drawn first clearly delineating

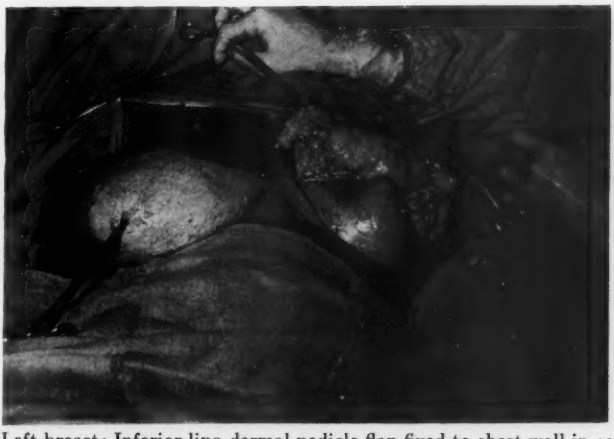

FIG. 6. Left breast: Inferior lipo-dermal pedicle flap fixed to chest wall in conical form. Superior flap will be superimposed.

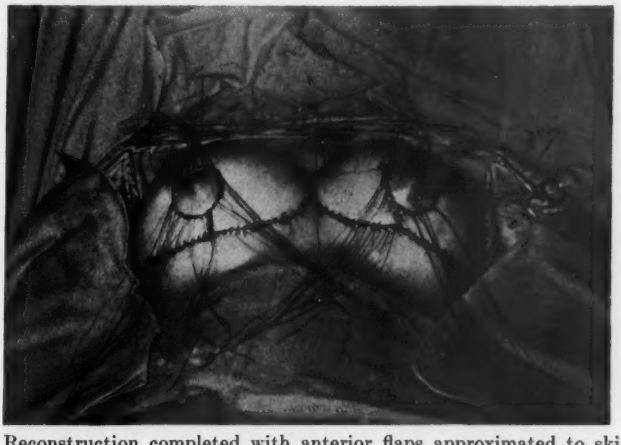

FIG. 7. Reconstruction completed with anterior flaps approximated to skin of chest wall. Nipple grafts stented in position ready for dressing.

the inframammary fold. With the breast in its usual pendulous position, a second line is marked out on the anterior surface; this parallels the inframammary line. These two lines intersect laterally in the axilla and medially in the region of the xyphoid.

The future areolar-nipple site is located on the midclavicular line approximately 21.5 cm. below the clavicle.[17] In selecting the new position of the nipple it is esthetically preferable to err on the low side; postoperatively, the apparent height of the nipple increases as the reconstructed breast becomes more pendulous. The midclavicular position is indicated on the inframammary line. While the breast is held in an extreme medial position, this latter point is joined by a vertical line to the center of the new nipple site; another vertical line connects

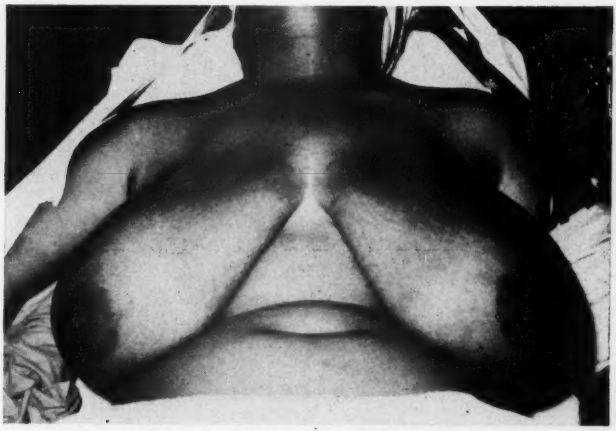

FIG. 8. Bilateral painful gigantomastia

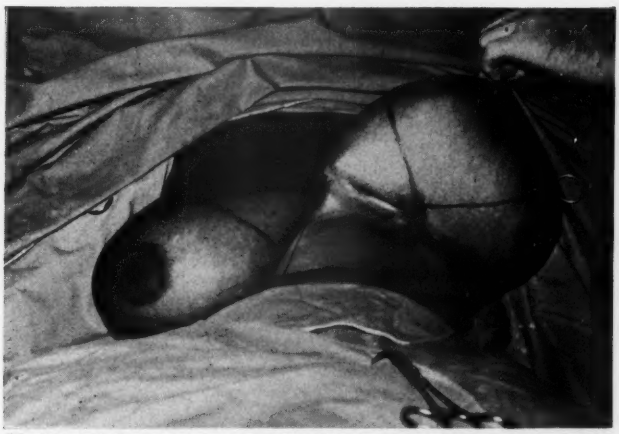

FIG. 9. Markings delineate upper anterior skin-fat flap which is to be used in reconstruction.

these same two points when the breast is displaced far laterally. Thus a triangle is formed with its apex at the future nipple site and its base on the line traversing the anterior surface of the breast and paralleling the inframammary line. This triangle represents a wedge resection which contributes to a more conical breast. A circle now is drawn using as an aid the ordinary 2-ounce medicine cup. Its circumference is within the areola. It has the nipple as its center (fig. 2).

Initially the nipple-areolar area with its smooth muscle is removed as a free full-thickness graft.[1] The entire surface of the breast inferior to the anterior transverse line then is denuded by removing a thick split skin graft (fig. 3). Next an incision is made traversing the anterior breast surface at the junction of intact skin and the lower dermal area. Through this incision a subcutaneous

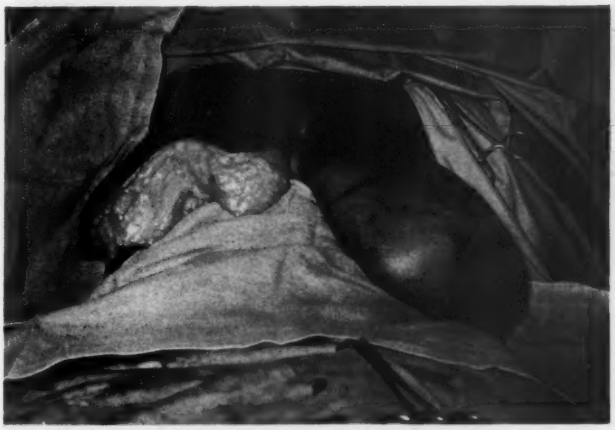

FIG. 10. All glandular tissue and lower portion of breast have been amputated

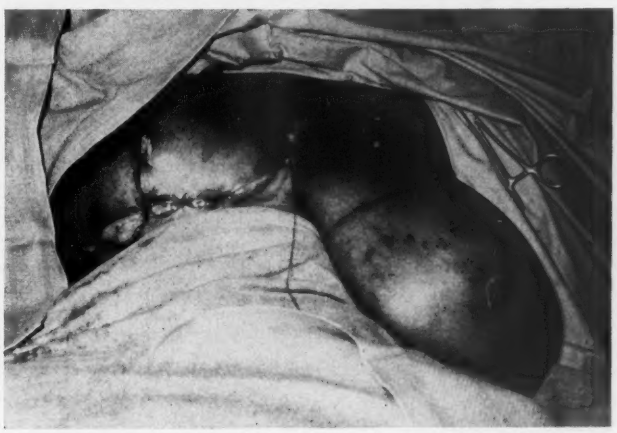

FIG. 11. Upper skin-fat flap closed over chest wall. Comparison of right and left breast shows the marked diminution in size which can be accomplished.

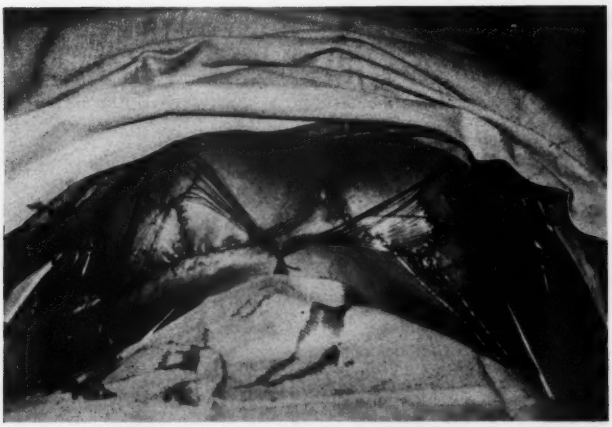

FIG. 12. Breast bilaterally reconstructed

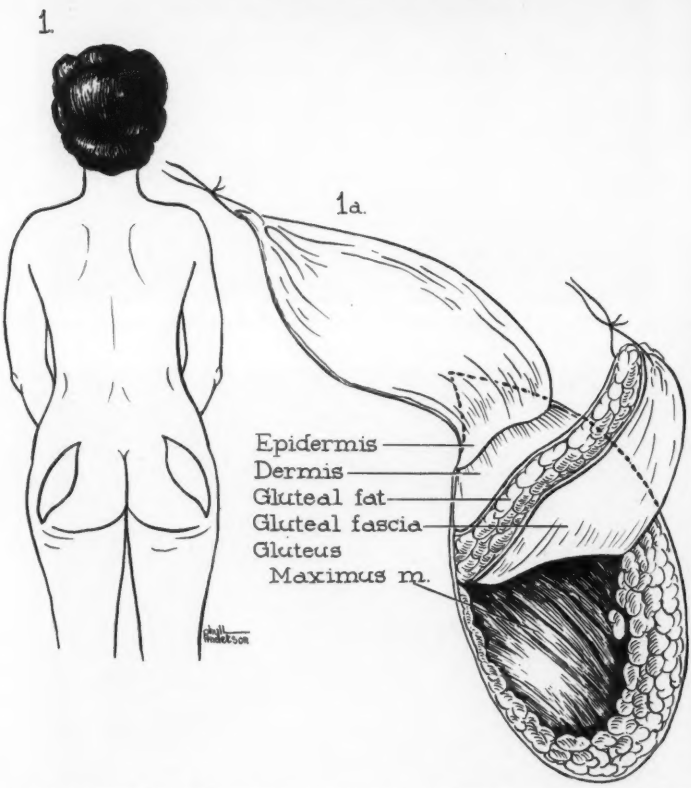

FIG. 13. (1) Illustrates donor site on buttocks for buttocks to breast transplant. (1a) Components of derma-fat gluteal fascia transplant.

resection of the mammary gland is done (fig. 4, right breast). The same incision defines the superior skin-fat flap and the inferior lipo-dermal pedicle flap (fig. 5). The inferior flap is fixed to the chest wall in a conical fashion at an appropriate level (fig. 6, left breast). A final superficial incision is made permeating the skin of the inframammary line. After inserting drains, the skin flap is sutured into position approximating the skin of the breast with that of the lower chest wall (fig. 4, left breast).

Finally the recipient area for the new nipple is prepared by removing all but the deeper dermal tissue from this site. The free graft then is carefully sutured and stented into position (fig. 7). A pressure dressing is applied to the reconstructed breast; a large size brassiere is used as the outer covering. Tape and other adhesive products thus can be avoided. The drains are removed in 48 hours. The nipple grafts are first dressed on the seventh day and all stitches are out on the tenth postoperative day[16]

The large breast composed of a great quantity of fatty subcutaneous tissue and a minimal amount of glandular element presents little problem (fig. 7). Aufricht's[2] analysis and reconstructive plan for pendulous breasts is applicable to this particular type of breast (fig. 8). The subcutaneous excision of the gland is carried out through an anterior incision. Since ample fatty substance is pres-

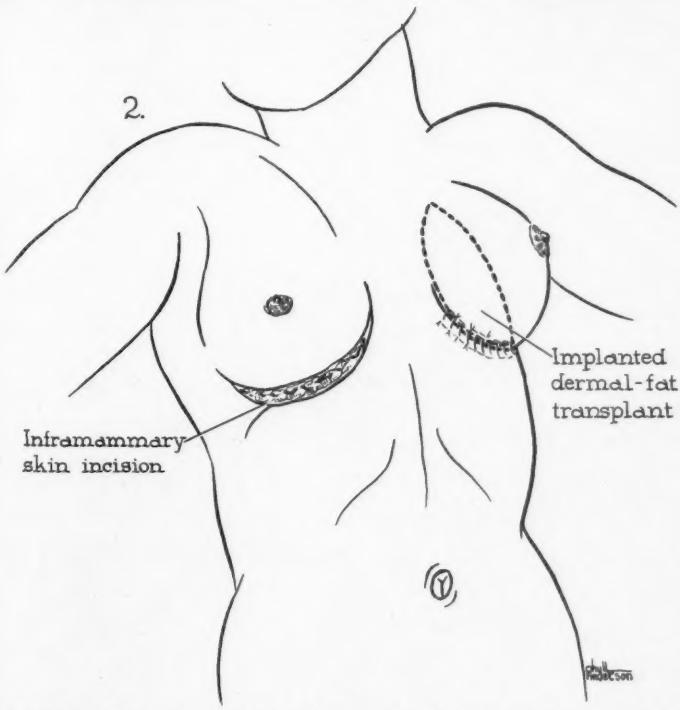

FIG. 14. Right breast flattened by subcutaneous simple mastectomy through inframammary incision. Left breast shows dermal-fat-fascia transplant in position.

ent on the superior flap, the entire lower portion of the breast may be removed with the gland (fig. 9). Skin edges are approximated (fig. 10). An areolar nipple graft completes the operation.[2, 3, 4] In the very large, pendulous breast, this resection and its reduction in breast bulk offers additional comfort (figs. 11, 12).

The small undersized breast with a preponderance of glandular element presents a more difficult restorative task. A variety of methods have been proposed for the cosmetic improvement of the small breast.[5, 10, 13] This problem is enhanced, moreover, by the subcutaneous simple mastectomy. A very direct approach to this reconstruction is based on Bame's procedure for the correction of the undersized breast.[6] After mastectomy through a submammary incision, a dermo-fat-fascia transplant from the buttocks can be inserted into the resulting breast defect. Thus breast elevation and contour are restored (figs. 13, 14).

SUMMARY

Total en bloc excision of the glandular portion of the breast and the restoration of acceptable breast contour may be accomplished in a single operative procedure. The reconstruction method employed depends upon the composition and size of the original breast. Lipo-dermal pedicle flaps, free nipple-areolar transplants, skin-fat flaps, and free dermo-fat-fascia transplants from buttocks to breast are utilized.

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